Anaesthesia

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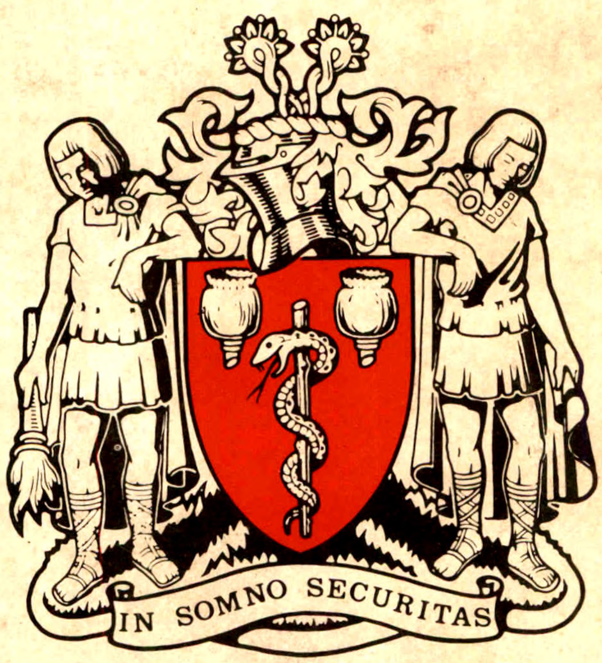

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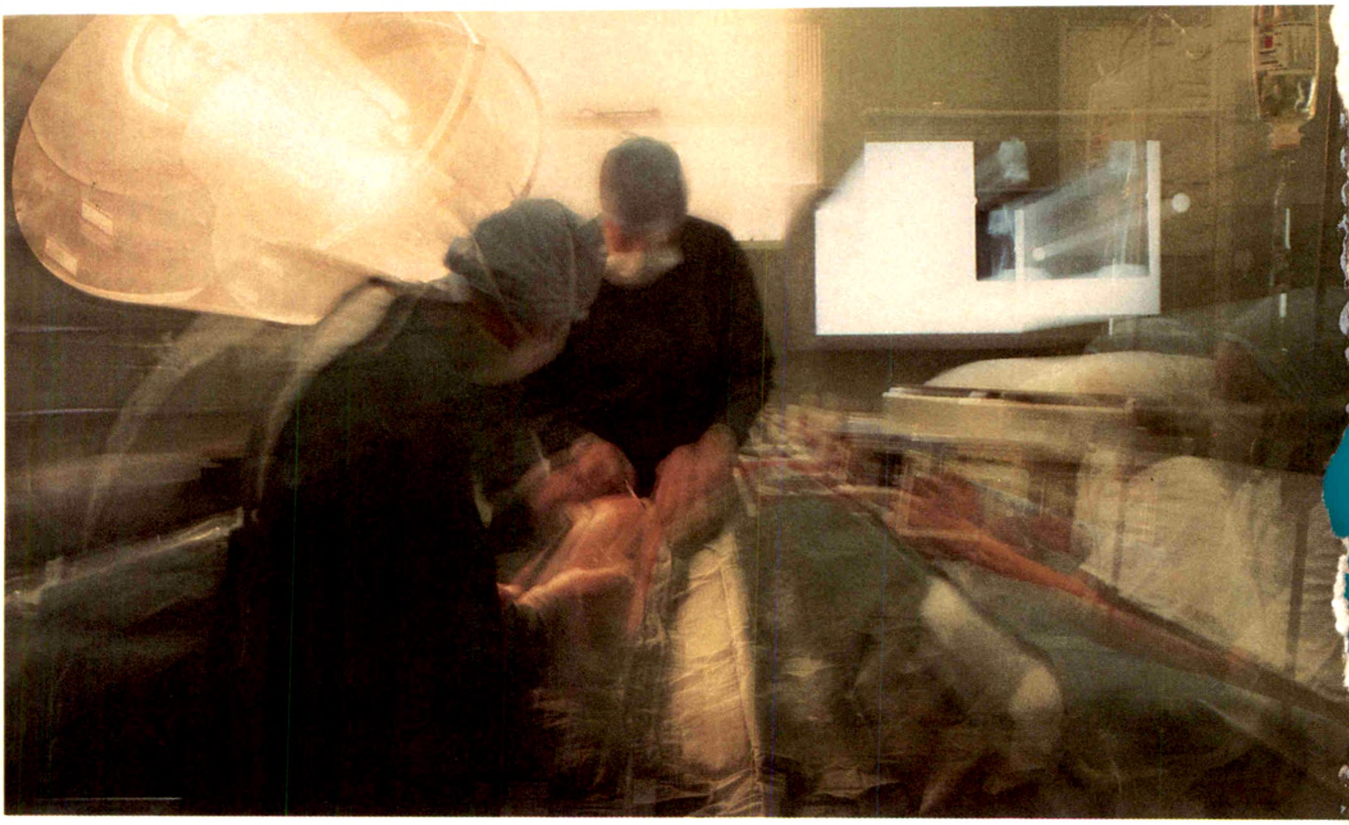

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Anaesthesia

Journal of the Association of Anaesthetists of Great Britain and Ireland

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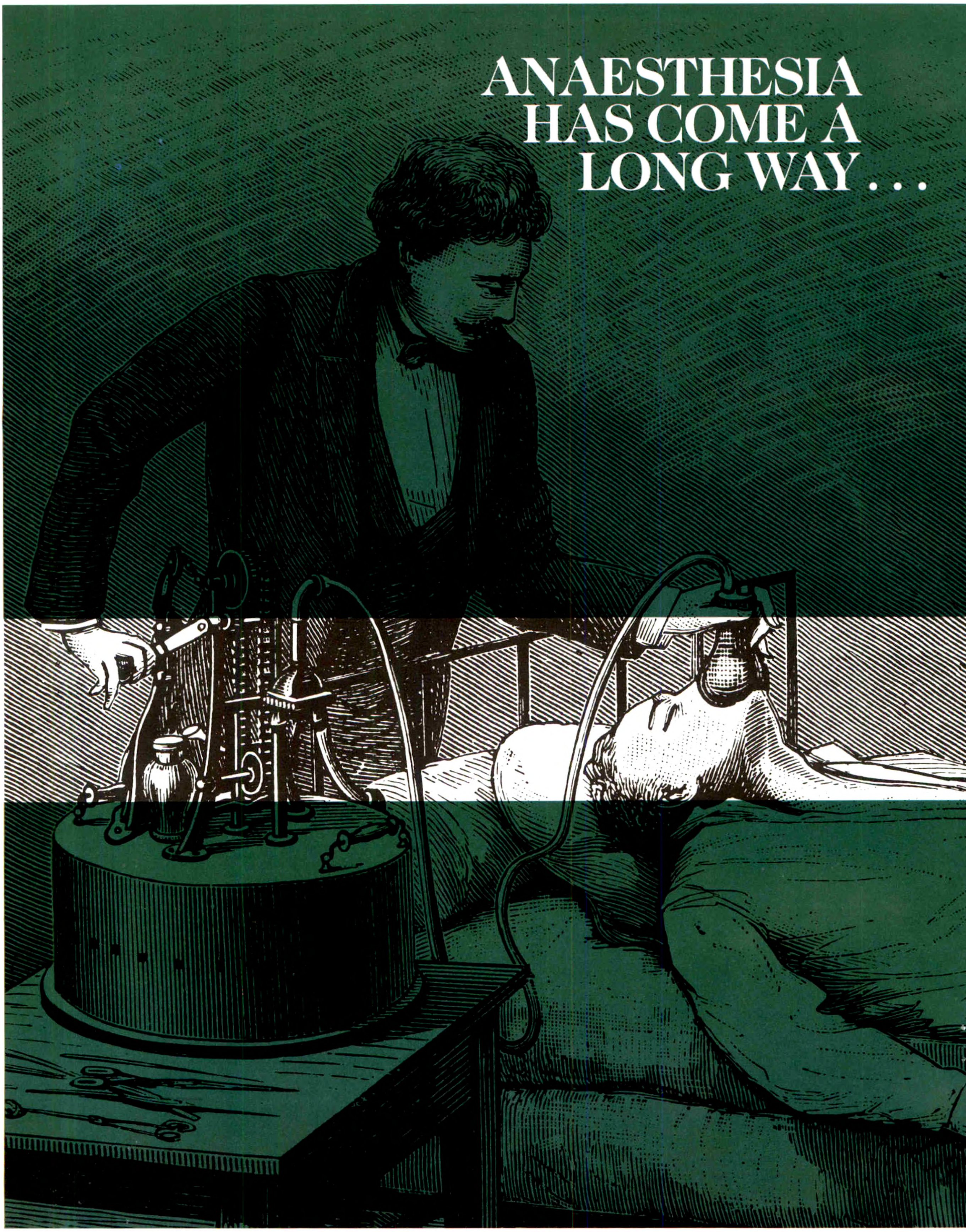

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Anaesthesia, 1989, Volume 44, page 803–804

Editorial

One more step?

There have been some important dates in the organisation of modern anaesthesia. In 1932, the Association of Anaesthetists of Great Britain and Ireland was founded at a meeting in London, with H.W. Featherstone as the first President. In 1947, the Faculty of Anaesthetists of the Royal College of Surgeons was formed after an initiative by the Association. A.D. Marston was the first Dean. In 1988, the College of Anaesthetists was born out of its 'parent', the Faculty, a step long-awaited and welcomed by anaesthetists everywhere. Michael Rosen is the first President.

The Association therefore welcomes its sturdy new grandchild, nearly one year old this month. The College takes its place among its fellow Colleges as an equal. We believe that outside bodies and individuals will at last begin to recognise that in the past our Faculty provided a first rate hospital inspection system and an examination, now in three parts, which is accepted as at least comparable with other higher medical qualifications.

The new College will continue to provide and develop the academic and educational functions of the Faculty. Though remaining within the building of the College of Surgeons, we believe that the independence of the College of Anaesthetists may be more clearly perceived on a much wider scale than previously.

There remains one further position to be achieved. It will not affect the educational role of the College, it will not improve the thoroughness of inspection visits to anaesthetic departments, it will not alter the representation of the College on the Joint Consultants Committee, the Standing Medical Advisory Committee or other representative bodies. The Faculty was already there. We expect to have the designation 'Royal'. The proper use of the title 'Royal' will finally demonstrate to all that anaesthesia, through the Faculty, has been an independent examining body for many years and has carried responsibility for the maintenance of standards within the Health Service. In the eyes of the public, anaesthesia will then rank alongside the other Royal Colleges, some of whom are considerably smaller than the College of Anaesthetists. This Association wholeheartedly supports the efforts of the President and Council of the College to have this title conferred, and to have it conferred without delay.

The Council of the College has invited the President of the Association to serve as a co-opted member of Council-in-committee. The President of the College and the Dean of the Irish Faculty of course sit on the Association Council. The Association is appreciative of the continued evidence of cooperation between Association and College and Faculty. In practice this has always been so. The two Councils agreed during the past year that there is a need for a major pump-priming input of money to improve the academic basis of intensive care. It was decided that this could best be achieved by funding a post of Senior Lecturer, with consultant status, in Intensive Care in an academic department of anaesthesia. The Association and the College would jointly fund this post for 5 years, with the expectation that the value of the post would be so evident from the contribution made by the incumbent that other sources of finance would enable the post to continue beyond the initial 5 years.

Academic departments were made aware of the availability of the funded post, and the joint steering committee received a number of worthwhile and detailed responses. The steering committee recommended, after visits to a short list of departments, that the Association and College Senior Lecturer in Intensive Care should be located at Birmingham University in the Queen Elizabeth Hospital. The post will be advertised at that hospital and it is anticipated that the appointment of a suitable anaesthetist will be made this autumn. This cooperative effort between the Association and College will be to the benefit of the specialty in years to come and might indeed encourage other benefactors to offer their support to intensive care. The Association and the College therefore come together for special projects, for example this Senior Lectureship and our regular joint Post Graduate Study Day held on a Saturday in November.

In general terms, however, the work of the Association and the College is complementary. The College is involved with supervision of training, examinations, education, maintenance of standards and indeed could be said to have a protective duty to the general public. These activities are all, of course, within the charitable status held by the College. The Association has a supportive role for its members, advises on contractual matters, concerns itself about standards of anaesthetic practice in the NHS and the private sector. Further, the Association makes recommendations on fees in the private sector, has educational and scientific responsibilities and provides resources for anaesthetic-related research. The Association also has international links with World and European anaesthesia and other national anaesthetic societies (we have had joint meetings with Danish, German and Canadian societies). Further the Association takes an interest in the anaesthetic problems of some Third World countries and has provided help for some British anaesthetists who both teach nationals and provide an anaesthetic service for a significant period in those countries.

The Association cannot have charitable status for help it gives to members and for matters concerning terms and conditions of service, but its education and research activities are properly granted charitable status.

Hundreds of members either write or visit 9 Bedford Square each year. The frequently held seminars have been an outstanding success and have allowed specialist topics to be investigated and discussed in a manner not possible in a larger scientific meeting. Over-subscribed seminars are often repeated and the seminar organiser is always ready to receive proposals for future meetings. These meetings (reachable within one day from anywhere in the British Isles) have become an important

feature of Association life. They have allowed members to explore a minority scientific interest or to pursue a health service topic.

Our large specialty (about one in eight consultants) needs to have its interests protected and its standards maintained. We are fortunate in being heirs to our colleagues of 1932 and 1947. They may not have foreseen all the changes that have taken place, but we, the inheritors, must ensure that the activities of our two professional bodies continue to lead in their field and be recognised by our members as fulfilling a perceived need.

Anaesthesia has been well served by having an Association and a College. Anaesthesia will be better served by having an Association and a Royal College.

Association of Anaesthetists of Great Britain and Ireland, 9 Bedford Square, London WC1B 3RA

M.M. Burrows
President

Editorial notices

Anaesthesia News

Copy for *Anaesthesia News* should be sent to the Editors as and when it becomes available. It is hoped that publication will occur within 2 months of receipt.

Manuscripts must be submitted in accordance with the internationally recognised *Uniform requirements for manuscripts submitted to biochemical journals* (*British Medical Journal* 1979: **1:** 432 5). Details will be foubnd in the Notice to Contributors to *Anaesthesia* at the end of this issue.

Anaesthesia, 1989, Volume 44, pages 805–807

Atracurium recovery: prediction of safe reversal times with edrophonium

J. N. CASHMAN, R. M. JONES AND A. P. ADAMS

Summary

The time to safe return of neuromuscular function after atracurium 0.3 mg/kg intravenously was assessed in 24 patients in whom anaesthesia was maintained with halothane 0.5%. Safe reversal (recovery to a T4 ratio > 0.5), after this dose of atracurium, could only be reliably and rapidly (< 2 minutes) achieved with edrophonium if a period greater than 30 minutes elapsed since administration of the relaxant. This coincides with the appearance of four recognisable twitches if a train-of-four pattern of nerve stimulation is used. Thus, if no monitoring equipment is available, at least half-an-hour should elapse after administration of atracurium in a moderate dose (e.g. 0.3 mg/kg) before rapid and reliable reversal can be anticipated. Four twitches in a train-of-four should be recognisable, if a nerve stimulator is available.

Key words

Antagonists; neuromuscular relaxants; edrophonium.
Monitoring; train-of-four stimulation.

A train-of-four ratio (T4 ratio) of 0.75 was shown to be associated with safe return of muscle power after spontaneous or evoked recovery from tubocurarine-induced neuromuscular blockade,[1] whereas after antagonism of atracurium a lower value of 0.5 may be considered safe.[2,3]

It is unlikely that the majority of anaesthetists will have access to the appropriate recording equipment necessary to determine quantitatively the T4 ratio, whether mechanomyographically or electromyographically. Visual and tactile evaluation therefore, remain the most likely means by which the majority of anaesthetists will assess recovery. However, it is visually difficult, even for experienced observers, to estimate correctly T4 ratio.[4,5] Tactile assessment of T4 ratio is little better,[5] although the addition of a preloading strain gauge may improve accuracy.[6] The time taken to attain T4 ratios of 0.5 and 0.75 from varying degrees of recovery, at end points readily quantified by visual and tactile means, may be clinically more useful.

This study set out to determine the average time taken to attain safe T4 ratios after reversal of atracurium-induced blockade at varying degrees of spontaneous offset. The overall objective of the study was to be able to recommend to those anaesthetists without access to sophisticated monitoring equipment, not just the average time after reversal of atracurium blockade of varying depth before safe return of muscle power is likely, but also the range (i.e. the minimum and maximum) of times likely to be encountered before safe reversal at any given degree of spontaneous recovery.

Patients and methods

The study was approved by the Hospital Ethics Committee, and 24 unpremedicated ASA class 1–2 patients of either sex who were scheduled to undergo general anaesthesia that required the use of a muscle relaxant, were studied. Their ages ranged from 18 to 39 years and their weights from 45 to 81 kg; no patient of greater than 20% above ideal weight was included in the study.

The nondominant arm of each patient was used for mechanomyographic monitoring, after it was first immobilised in a Stanec splint.[7] Muscle paralysis was assessed by stimulating the ulnar nerve via surface electrodes, using a train-of-four pattern of supramaximal 0.2 msec duration square wave impulses at 2 Hz, repeated every 12 seconds (Myotest). The evoked force of contraction of the *adductor pollicis* muscle was measured by a miniature force displacement transducer (FDT-10; 0–10 kg range) connected to a Devices pre-amplifier and chart recorder. Twitch height was recorded during onset, spontaneous offset and after

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An abstract of this work was presented at the 10th Annual Meeting of the European Academy of Anaesthesiology, September 1988.

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edrophonium administration. Only records in which, after evoked reversal, T1 had returned to within 90–110% of control were considered as suitable for analysis.

Anaesthesia was induced with thiopentone 4–6 mg/kg, after which the patient breathed 66% nitrous oxide in oxygen with 1% inspired halothane, delivered by a Bain type coaxial breathing system and facemask. Baseline neuromuscular recordings were obtained and, when stable, atracurium 0.3 mg/kg was injected intravenously as a rapid bolus followed by 1 ml of normal saline. The trachea was intubated when the first twitch of the train (T1) was 10% of control or less, after which halothane was reduced to 0.5% and ventilation controlled to maintain an end-tidal CO_2 of between 4.5 and 5.5 kPa. Residual neuromuscular blockade was antagonised at the end of surgery by edrophonium 0.5 mg/kg together with atropine either when there was no measurable twitch in response to stimulation, (T1: Control 0%, six patients), or when the first twitch (T1) had returned to 25% of its control value (T1: Control 25%, six patients); to 50% of its control value (T1: Control 50%, six patients); to 75% of its control value (T1: Control 75%, six patients). The times taken to achieve a T4 ratio of 0.5 and of 0.75 were recorded.

All demographic data are reported as mean (SD) whilst remaining data are expressed as median values together with ranges where appropriate.

Results

Twenty-nine patients were studied, but the records of only 24 were suitable for analysis based on the described criteria. The demographic data of the resultant four groups are outlined in Table 1.

Table 1. Demographic data, mean (SD).

	Age (years)	Weight (kg)	Sex
T1: Control 0%	24.7 (2.6)	59.0 (5.7)	6F
T1: Control 25%	26.2 (4.8)	61.8 (11.1)	4F/2M
T1: Control 50%	22.8 (4.0)	65.8 (12.1)	3F/3M
T1: Control 75%	21.2 (2.8)	62.7 (5.4)	2F/4M

During onset, the median time to achieve a T1 which was 10% of control was 2.5 minutes (range 1.3 to 9.0 minutes; all patients). The median duration of block prior to reversal in the T1: Control 0% group was 20.1 minutes (range 15.6 to 25.0; $n = 6$); in the T1: Control 25% group, 30.8 minutes (range 26.3 to 38.1; $n = 6$); in the T1: Control 50% group, 31.3 minutes (range 25.8 to 38.3; $n = 6$) and in the T1: Control 75% group 40.5 minutes (range 34.3 to 63.6; $n = 6$). The T4 ratios at reversal are shown in Table 2. When T1 had returned to 50% of control, four twitches were always present in the train-of-four. The times to achieve a T4 ratio of 0.5 and 0.75 are shown in Table 3.

Table 2. T4 ratios at reversal.

	T4 ratio, median (range)
T1: Control 0%	0
T1: Control 25%	12 (0–17)
T1: Control 50%	15 (10–19)
T1: Control 75%	36 (19–47)

Table 3. Offset times (minutes).

	Median times and ranges for evoked recovery to a T4 ratio of	
	0.5	0.75
T1: Control 0%	12.3 (6.5–18.8)	19.5 (11.5–24.2)
T1: Control 25%	1.2 (0.5– 2.2)	5.6 (0.9–15.2)
T1: Control 50%	0.7 (0.4– 1.2)	1.1 (0.7– 4.0)
T1: Control 75%	0.4 (0.1– 0.6)	0.6 (0.2– 0.8)

Discussion

Reversal of atracurium-induced neuromuscular blockade of less than 25 minutes duration was prolonged in this study of patients in whom anaesthesia was maintained with halothane 0.5%. The median time for recovery to a T4 ratio of 0.5 was 12.3 minutes and to a T4 ratio of 0.75 was 19.5 minutes, whilst the longest times taken were 18.8 and 24.2 minutes respectively. For anaesthesia that lasted more than 25 minutes, some spontaneous recovery had always occurred prior to evoked reversal. Thus for a T1 25% of control a T4 ratio of 0.5 was achieved in 1.2 minutes; however, recovery to a T4 ratio of 0.75 was still slow, and took a median of 5.6 minutes. The longest time taken was 15.2 minutes. However, two patients did not have all four twitches present to train-of-four stimulation and, if these are excluded, the median recovery times (to T4 ratios of 0.5 and 0.75) become 0.9 and 3.8 minutes respectively; the longest was 11.3 minutes. Evoked recovery at a T1, which was 50% of control, was associated with rapid recovery, both to a T4 ratio of 0.5 (0.7 minutes) and 0.75 (1.1 minutes); the slowest was 7 minutes. A T1 75% of control was invariably associated with recovery in less than a minute. All four twitches to train-of-four stimulation were present in both these groups.

It was previously reported that a T4 ratio 0.5 is associated with safe return of muscle power after atracurium- or vecuronium-induced blockade.[2] This view was supported by Astley and Payne.[3] However, it may be that the rate of evoked recovery from 0.5 to 0.75 after such neuromuscular blocking agents is so rapid that stopping anaesthesia at a T4 ratio of 0.5 is associated with a T4 ratio of 0.75 by the time of extubation. Jones *et al.* in their study observed patients for 3 hours and found no evidence of recurarisation using standard tests such as head lift,[2] whilst Astley and Payne also found no evidence of recurarisation, even though monitoring was continued for a further 15 minutes.[3] In this study, the median time from a T4 ratio of 0.5 to one of 0.75 was 5.5 minutes (T1: Control 0%), 2.9 minutes (T1: Control 25%), 0.3 minutes (T1: Control 50%) and 0.2 minutes (T1: Control 75%). These results lend support to our theory, but only in circumstances when some spontaneous recovery has occurred. In those patients in whom no spontaneous recovery has occurred, current evidence suggests that neostigmine, a drug with a 'slower' onset, should be preferred to edrophonium.[2,3,8]

A number of studies have attempted to correlate visual and tactile assessment of T4 ratio with mechanical and EMG measurement. Both Savarese and Ali[1] as well as Viby-Mogensen[4] previously showed that it is difficult to estimate the degree of fade in train-of-four with visual and tactile methods. Foldes, in an attempt to overcome some of the shortcomings of clinical assessment, has developed a

simple device called the Myoscan which consists of a preloading spring for attachment to the thumb.[6] Stimulation of the ulnar nerve elicits visible expansion of the spring. The Myoscan seems to be the most useful clinical method so far developed, but even this method appears to underestimate the degree of residual paralysis.[5]

The ability to be able to predict times to safe reversal has obvious attractions in the absence of mechanomyographic or electromyographic monitoring. Thus, four twitches should always be present prior to reversal. However, in view of the slow recovery from a deep block, we suggest that in cases where surgery was unexpectedly rapid and a nerve stimulator is not readily available, it is worth waiting 30 minutes after a dose of atracurium 0.3 mg/kg before attempting reversal. Thus, although atracurium is one of the newer generation of 'intermediate duration' non-depolarising muscle relaxants it should be emphasised that even after a minimum intubating dose (0.3 mg/kg) evoked reversal should not be attempted until at least 30 minutes has elapsed from the time of administration.

In conclusion, recovery to a T4 ratio of 0.5 occurred in less than 2.2 minutes in all patients in whom T1 was greater than 25% of control, and to a T4 ratio of 0.75 in less than 4 minutes in all patients in whom T1 was at least 50% of control. In the absence of monitoring equipment, about 30 minutes should have elapsed after an intubating dose of atracurium (0.3 mg/kg) and (or) four twitches should be visible, before attempting to evoke recovery, if reversal is reliably to be achieved in less than 5 minutes.

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Anaesthesia, 1989, Volume 44, pages 808–811

Gastro-oesophageal reflux in pregnancy at term and after delivery

R. G. VANNER AND N. W. GOODMAN

Summary

Gastro-oesophageal reflux during a 40-minute reflux provocation test was assessed by lower oesophageal pH monitoring in 25 pregnant women at term, and again on about the second day after delivery. At term 17 women refluxed a total of 29 times; after delivery five women refluxed once each. There was a significant decrease in gastro-oesophageal reflux by the second day after delivery ($p < 0.05$). Gastro-oesophageal reflux is, however, only one of the factors that predisposes to acid aspiration pneumonitis.

Key words

Complications; aspiration pneumonitis, gastro-oesophageal reflux.
Anaesthesia; obstetric.

Pregnant women at term who need a general anaesthetic must be protected against the risks of regurgitation and possible aspiration pneumonitis. Gastro-oesophageal reflux increases these risks; symptoms of reflux occur in 80% of women at term,[1] and lower oesophageal pH monitoring has shown an increased incidence of reflux at term, even in asymptomatic women.[2,3]

There is no clear advice about when these women can be treated as normal after delivery, but precautions against regurgitation and aspiration pneumonitis, continued unnecessarily, have their own risks. In this study we used lower oesophageal pH monitoring to compare gastro-oesophageal reflux at term with that on the second day after delivery. A preliminary report has been presented.[4]

Method

Ethical permission was obtained for the study. Informed consent was given by 28 of the 85 mothers who were asked, a rate of recruitment of about 35%. Twenty-five of the 28 women were studied both before and after delivery; 16 were primigravida and nine multigravida. All but three had symptoms of gastro-oesophageal reflux at term; none had had symptoms before pregnancy, and none had symptoms more than 12 hours after delivery. They were starved for 4 hours and did not smoke for 2 hours before the study. No medication was taken that affects gastro-intestinal function.

Insertion of electrode

pH was measured with a monocrystalline antimony electrode[5] mounted in a soft plastic catheter of 2 mm diameter (Synectics Medical). The electrode was cleaned with alcohol and calibrated with buffers at pH 7 and pH 1 before each use. One nostril was anaesthetised with lignocaine, while the patient sat. The electrode was inserted and swallowed with 200 ml of water. The gastric pH was measured before the electrode was withdrawn to the gastro-oesophageal junction, identified by a sudden increase in pH. The final position of the electrode was 5 cm above that point. The electrode was well tolerated by all the patients.

Reflux test

A reflux episode was defined as a decrease in lower oesophageal pH from the normal value of about 6 to below 4. The duration of the episode was taken as the time for which the pH was below 4. The mothers were conscious and studied in four positions: supine with 15° left tilt, left lateral, right lateral, and lithotomy with 15° left tilt. The patient carried out a sequence of manoeuvres designed to provoke reflux if there was no spontaneous reflux in 5 minutes: they were asked to cough, to strain (Valsalva), and then to sniff with the nose pinched (Müeller). The oesophageal pH was allowed to return to normal before the patient assumed the next position, if there was reflux; this gave a possible maximum of four refluxes during one reflux

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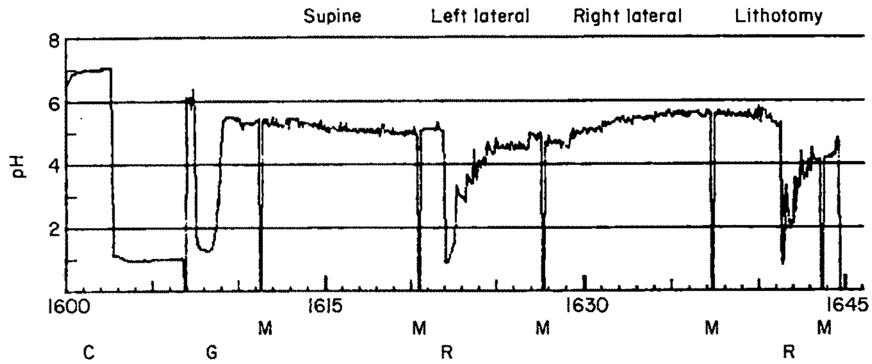

Fig. 1. Oesophageal pH in a pregnant woman at term. C: calibration of the electrode at pH 7 and pH 1; G: gastric pH as the electrode is inserted into the stomach before withdrawal to the oesophagus; M: a marker to indicate changes of position; R: clear episodes of gastro-oesophageal reflux, oesophageal pH is the same as gastric pH. Each reflux episode lasts for about 2 minutes.

test. This reflux test is similar to Skinner's Standard Acid Reflux Test,[6] but he used 20° head-down tilt, not the lithotomy position. The same test was used for all 25 pregnant women beyond 36 weeks of gestation and then again at about 36 hours after delivery. The pH data were stored by a digital recording device every 4 seconds and analysed later by a computer program (Digitrapper and Oesophogram, Synectics Medical).

Results

A graph of oesophageal pH against time was produced for each reflux test. Oesophageal pH decreased to gastric pH, if reflux occurred, and then increased, typically stepwise, as the oesophagus was cleared (Fig. 1).

Seventeen out of 25 mothers had gastro-oesophageal reflux at term, and between them a total of 29 reflux episodes was seen. Five out of 25 mothers had gastro-oesophageal reflux after delivery; each had one episode of reflux (Fig. 2). The decrease from 17 to 5 is significant ($p < 0.05$ Chi-squared test with Yates' correction; 95% confidence limits on postdelivery incidence of reflux 0–11). Table 1 shows the time after delivery at which each woman was studied.

Some of the refluxes were shorter. Three of 29 at term and three of five after delivery lasted less than 12 seconds, and they appeared as spikes on the graph (Fig. 3). Both the women who after delivery had single refluxes longer than 12 seconds were tested within 30 hours of delivery and had had more than one reflux when tested at term.

The positions in which the 29 refluxes occurred, during the reflux tests at term, are listed in Table 2. Of the 29 refluxes, 16 were spontaneous and 13 provoked. The provoking manoeuvres that caused these 13 reflux episodes are listed in Table 3. The patients complained of heartburn during 20 of the 29 refluxes. The three women who had no symptoms of gastro-oesophageal reflux during pregnancy all refluxed once at term. These refluxes were asymptomatic. The mean pH of the gastric contents before delivery was 1.42 (range 0.8–2.6) and after delivery 1.46 (range 0.6–3.0).

One mother who refluxed three times at term but did not reflux during the test 34 hours after delivery, agreed to have prolonged ambulatory pH monitoring. An indication of the amount of gastro-oesophageal reflux is given as the percentage of time that the pH was below 4: for 24 hours at term 2.8%, between 12 and 24 hours after delivery 2.0%, between 24 and 36 hours after delivery 1.2%, and for 24 hours 11 weeks after delivery 3.6%.

Discussion

Pregnant women at term have more gastro-oesophageal reflux during provocation tests than do normal controls.[2,3] Our results show that the incidence of reflux decreases by the second day after delivery; and three of the five women who did reflux at this time did so for less than 12 seconds.

Acid gastric contents are cleared from the oesophagus by primary peristalsis (swallowing) and secondary peristalsis (local reflex), as well as by the neutralising effect of saliva.[7,8]

Table 1. Gastro-oesophageal reflux and the time after delivery.

	Patient																								
	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25
Reflux episodes at term	2	2	0	1	0	2	3	0	1	2	1	1	3	3	1	0	0	2	0	0	1	2	0	1	1
Reflux episodes after delivery	0	1	1*	0	0	1	0	0	0	1*	0	0	0	0	0	0	0	0	1*	0	0	0	0	0	0
Hours after delivery	24	26	26	28	29	30	30	30	30	34	34	34	34	35	36	36	36	38	45	46	51	55	64	66	80

*Indicates a spike reflux after delivery.

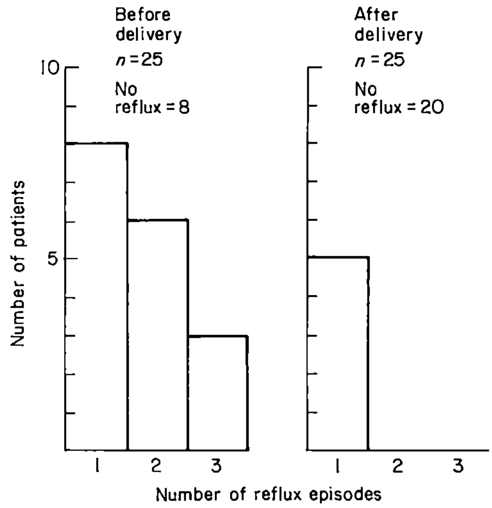

Fig. 2. The number of women who had 0, one, two or three reflux episodes during the reflux test at term and after delivery. No patients had the maximum of four reflux episodes.

Table 2. Position and reflux at term.

Position	Number of refluxes in each position
Right lateral	10
Left lateral	8
Lithotomy	6
Supine	5
Total	29

Clearance of acid causes a stepwise increase in lower oesophageal pH, to give an average duration of reflux of 60 seconds in this study. Spike refluxes (Fig. 3) probably occur when the oesophagus is cleared rapidly by a secondary peristaltic sequence.[7] This is a protective role of the oesophagus which decreases the duration of reflux and lessens the chance of regurgitation. However as both sleep[9] and general anaesthesia[10] can inhibit oesophageal motility, the rapid clearance seen in these short refluxes in conscious patients may not occur during anaesthesia. Twenty-six of Skinner's 91 normal subjects[6] showed similar short refluxes. These spike refluxes are probably normal events because the two reflux tests are similar. Two of our 25 women did have frank reflux after delivery; these two were tested relatively early after delivery and both showed more reflux at term than after delivery.

Recently, the mechanism of reflux was studied by the continuous measurement of lower oesophageal sphincter pressure combined with lower oesophageal pH monitoring.[11,12] They showed that most refluxes are caused by transient relaxations of the lower oesophageal sphincter. Provoking manoeuvres that raise gastric pressure above the pressure of the lower oesophageal sphincter, which may itself be decreased, cause few refluxes. These transient relaxations are neurally mediated,[13] initiated by gastric distension,[14] and are inhibited by anaesthesia.[15] This study supports this mechanism of gastro-oesophageal reflux since over half the reflux episodes occurred spontaneously with the patient lying quietly.

Most operations that require general anaesthesia after delivery are performed in the lithotomy position. The highest intragastric pressures occur in this position at term,[16] and more gastro-oesophageal reflux might be expected. There was no obvious effect of position on the incidence of reflux in this study.

Previous studies using lower oesophageal pH monitoring show that pregnant women at term have an increased incidence of gastro-oesophageal reflux even if they do not have symptoms,[2,3] although these workers did not report whether the reflux episodes were symptomatic. Our three asymptomatic women did not complain of heartburn even during gastro-oesophageal reflux; perhaps they did not have an acid sensitive oesophagus.

Gastro-oesophageal reflux decreased progressively in the 36 hours after delivery in the mother who had prolonged ambulatory lower oesophageal pH monitoring, which seems to confirm the results of the reflux tests. She had more gastro-oesophageal reflux than at term in an attempted baseline period 11 weeks after delivery. This was unexpected but was not a true baseline since she was at home and more active than in hospital.

The plasma progesterone concentration decreases to concentrations similar to those in the luteal phase of the menstrual cycle within 24 hours of delivery.[17] The relaxant effect of progesterone on the smooth muscle of the lower oesophageal sphincter may be a cause of the increased incidence of reflux during pregnancy.[18] Reflux can also be caused by gastric distension[14] if gastric emptying is delayed. Pregnant women have a delay in gastric emptying[19] which may also be caused by progesterone. However, Blouw *et al.*[20] showed that the volume and pH of

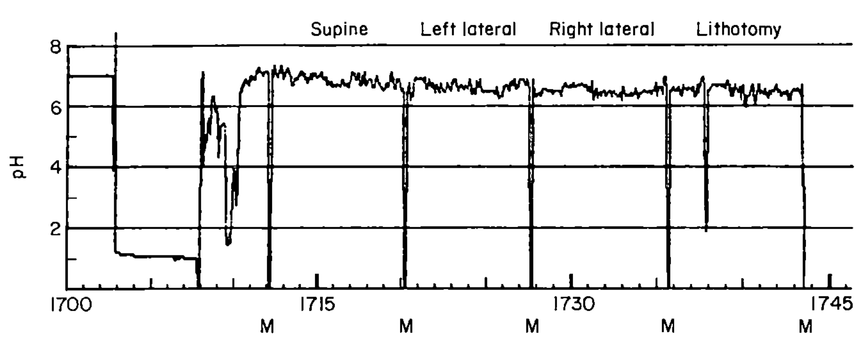

Fig. 3. Oesophageal pH in a woman after delivery. The sequence of calibration, placement and the reflux test is as in Fig. 1. M: a marker to indicate changes of position. A reflux episode occurred in the lithotomy position that lasted for 8 seconds and appears as a spike on the graph.

Table 3. Provoked reflux at term.

Manoeuvre	Number of refluxes provoked by manoeuvres
Turning	7
Valsalva	3
Mueller	2
Cough	1
Total	13

gastric contents were no different after a period of starvation within 48 hours after delivery than they were in patients who had not recently been pregnant. These points support our observation that the incidence of gastro-oesophageal reflux decreases by the second day after delivery.

Pregnant women at term may have an increased interstitial lung water; this makes the lungs more susceptible to pneumonitis after aspiration of gastric contents.[21] Lung water has not been measured during pregnancy, but there is an increase in closing volume, and a proportion of women have changes consistent with interstitial pulmonary oedema on a chest radiograph, taken soon after delivery.[22] There is a considerable increase in extracellular fluid volume during pregnancy, which increases body weight. Body weight remains high for the first 3 days after delivery, before a diuresis causes a rapid decrease in weight over the next 3 days.[23] Weight loss is more gradual by the seventh day, and by then the diuresis is complete, but lung water has not been measured at this time.

Even though the high incidence of gastro-oesophageal reflux at term approaches normal by the second day after delivery, other factors are involved so it is not yet possible to make a clear statement about when specific precautions against regurgitation and acid aspiration can safely be discontinued after delivery.

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Premedication with temazepam in minor surgery

The relationship between plasma concentration and clinical effect after a dose of 40 mg

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Summary

Fourteen patients received oral premedication of temazepam in soft gelatin capsules before minor surgery. The plasma concentrations of temazepam and its sedative, anxiolytic and amnesic effects were measured for 24 hours. Absorption was rapid and peak concentrations occurred 49 minutes after administration. Clinical effects were evident at 30 minutes and persisted for about 4 hours. The decline in plasma concentration was biexponential with a distribution half-life of 1.24 hours. The end of the distribution phase coincided approximately with the termination of its clinical effects. A relationship between plasma concentration and effect was observed; concentrations above 300 ng/ml produced measurable changes in tests of mental function. Patients had recovered fully from the effects of temazepam after 24 hours. This dose of temazepam is reliable and effective as premedication before surgery.

Key words

Premedication; temazepam.

Temazepam (3-hydroxydiazepam) is absorbed well and has a rapid onset of action when administered orally in soft gelatin capsules.[1,2] Unlike diazepam, it is metabolised rapidly, principally by conjugation, to pharmacologically inactive metabolites.[3] Its short half-life and its sedative and anxiolytic properties make it an effective premedicant when given before anaesthesia for minor surgical procedures.[4,5] Most studies have reported on the effects of 20 mg or 30 mg doses of temazepam[6,7] but a 40-mg dose was found to be an effective alternative to intravenous diazepam (Diazemuls) 10 mg for sedation during removal of impacted third molar teeth.[8]

The purposes of the present study were to obtain clinical and plasma concentration data during the first 4 hours after oral administration of temazepam 40 mg, to assess the extent of intersubject variability and to correlate plasma concentrations with clinical effects. Clinically effective plasma concentrations of temazepam could thus be established. In addition, residual clinical effects and plasma concentrations were measured after 24 hours to provide information on the duration of action at this dose.

Patients and methods

Informed consent was obtained from 14 nonambulatory inpatients of ASA grades 1 or 2 scheduled for minor elective surgery, after ethics committee approval. Nine males and five females, who were aged between 22 and 59 (mean 42) years and who weighed between 55 and 87 (mean 69) kg participated in the study. None had received recent regular drug medication or psychotropic therapy. Patients who were pregnant and those suffering from asthma or drug hypersensitivity were excluded from the study.

An oral dose of four 10-mg soft gelatin temazepam capsules (Normison, Wyeth) was administered with 30 ml water 4 hours before surgery. Samples of venous blood (5 ml) were collected from each patient in heparinised containers before and 0.5, 1, 1.5, 2, 3, 4, 9 and 24 hours after drug administration. An indwelling intravenous cannula (16 or 18 G) flushed with heparinised saline (10 units/ml) was used throughout the procedure. The blood samples were centrifuged immediately and the plasma separated and stored at −20°C. Plasma temazepam concentrations were determined by gas chromatography using a Pye-Unicam chromatograph fitted with a ^{63}Ni electron capture detector. The method used was essentially that described by Belvedere *et al.*[9] except that BSTFA (N, O-bis [trimethylsilyl] trifluoroacetamide) was used to silylate the dried ethereal extracts. Diazepam was added to the plasma as an internal standard before extraction and all the results were averaged from analyses performed in duplicate.

The amnesic, sedative and anxiolytic effects of tem-

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Table 1. Plasma concentrations (ng/ml) of temazepam after an oral dose of 40 mg.

	Time (hours)*							
Subject	0.5	1	1.5	2	3	4	9	24
1	1400		750	406		371	92	26
2	1740	1422	1317	1020		709	227	47
3	1890	1737	1180	867		388	161 (10)	28
4	1655	1465	1114	1088	865	618	145	43
5	1393	1432	1697	1897	815	561	376	191
6	650	851	758	604	345	292	100	25
7	1384	1683	1297	993	798	670	217 (7)	26 (22)
8	1807	1133	1011	611	281	201 (3.5)	68	34
9	1533	965	957	807	445	386 (3.5)	53 (13)	21
10	1737	1123	957	652	574	433	207	50
11	1733	1267	1079	797	582	459		30
12	1016	1323	1070	767	445	300	191 (6)	32
13	872	1890	1744	733	458	347	176	29 (22)
14	581	643	701	576	443	409 (3.5)	172	30

*Time for some samples varied from the column headings. In these cases the time is given in parentheses after the plasma concentration.

azepam were assessed in each patient during the first 4 hours after administration at the same times as specified for blood sampling. Anxiety was scored using a 100-mm visual analogue scale and sedation was scored on a five-point scale. These tests, together with those for short-term recall and for retrograde and anterograde amnesia, have been described fully in the report of an earlier study.[10] In addition, the patients were questioned about their well-being and location. If the patient was too sleepy to reply, this was recorded also. At 0 and 24 hours, the digit symbol substitution test and tests for logical memory and mental control[11] were carried out. The same observer conducted all the assessments of an individual patient.

Wherever possible, anaesthesia was standardised using thiopentone 3–5 mg/kg, fentanyl 1 μg/kg, no less than 30% oxygen in nitrous oxide and enflurane 1–3%. Atracurium 0.5 mg/kg was administered if muscle relaxation was required. Papaveretum 15–20 mg and prochlorperazine 12.5 mg were given intramuscularly as required after operation.

The clinical data obtained during the first 4 hours were analysed by Chi-square test (amnesia and short-term recall) and the Wilcoxon matched-pairs signed-ranks test (sedation and anxiety). The Mann-Whitney *U* test was used to test for significance at different plasma temazepam concentrations and the Wilcoxon matched-pairs signed-ranks test was used to compare the results of tests carried out before treatment with those after 24 hours.

Results

Plasma temazepam concentrations and pharmacokinetic data

Table 1 shows the plasma temazepam concentrations in all 14 patients. Absorption of temazepam was rapid; peak concentrations were achieved within 30 minutes in eight subjects and within 120 minutes in all subjects. Temazepam concentrations in excess of 500 ng/ml were observed in all patients within 30 minutes. The mean (SD) peak plasma concentration was 1560 (375) ng/ml and this was attained 49 (28) minutes after administration.

The decline in plasma concentration was biphasic. The results from each patient fitted with a two-compartment model and the distribution and elimination half-lives of temazepam were calculated by the method of residuals. The mean (SD) α (distribution) half-life was 1.24 (0.50) hours

Table 2. Clinical effects of oral temazepam 40 mg.

Time (hours)	0 ($n = 14$)	0.5 ($n = 14$)	1 ($n = 14$)	1.5 ($n = 14$)	2 ($n = 14$)	3 ($n = 10$)	4 ($n = 13$)
Recall of memory cards after 24 hours (%)	100	35.7*	28.6*	42.9*	35.7*	80.0	38.5*
Recall and recognition of memory cards after 24 hours (%)	100	71.4	42.9*	78.6	78.6	90.0	84.6
Short-term recall (%)	100	50.0 *	35.7*	42.9*	71.4	60.0.	61.5*
Sedation score median	1	2*	3*	2*	2*	3*	2*
Interquartile range	0	1	1	1	1	1	1
Anxiety score median	35	14	13*	12*	14.5	20.5	18
Interquartile range	71	33	24	17	25.5	30.5	39.5

*Significantly different from time = 0, $p < 0.05$.

Table 3. Relationship between plasma concentration of temazepam and clinical effect.

	Plasma concentration (ng/ml)				
	0–300	301–600	601–900	901–1200	1201+
Recall of memory cards after 24 hours (%)	88.9+	64.7+	40.0*	42.9*	26.1*
Recall and recognition of memory cards after 24 hours (%)	99.4+	88.2+	85.0+	78.6	53.8*
Short term recall (%)	94.4+	58.8*	60.0*	64.3	34.8*
Sedation score median	1+	2*+	2*+	2*+	3*
Interquartile range	1	1	0.5	1	2
Anxiety score median	35+	29+	27+	12.5*	7*
Interquartile range	57	38	24	14	12

*Significantly different from Group 0–300, $p < 0.05$.
+Significantly different from Group 1201+, $p < 0.05$.

and the mean β (elimination) half-life was 7.5 (2.8) hours. However, the latter value is unreliable because insufficient measurements of plasma concentration were made after the first 4 hours. At 24 hours, plasma concentration exceeded 50 ng/ml in only one patient, the oldest in the study (subject 5). In this patient (a female who weighed only 56 kg), absorption of temazepam was the slowest and the peak concentration was the highest observed in the entire study.

Clinical effects

The clinical effects during the first 4 hours after administration of the 40-mg dose are summarised in Table 2. The maximum effects of temazepam on memory, sedation and anxiety occurred after one hour when most of the patients were asleep but easily rousable. The effects persisted throughout the observation period and although some recovery occurred, memory and sedation were still significantly different from normal after 4 hours.

The sedative, anxiolytic and amnesic effects were assessed in the individual patients at the same times that blood samples were withdrawn for analysis. Thus, it was possible to explore the relationship between plasma temazepam concentration and clinical effect. These relationships are shown in Table 3, in which the temazepam concentrations at each assessment time are grouped at 300 ng/ml intervals and the corresponding clinical effects shown. Concentrations of temazepam above 300 ng/ml affected memory and wakefulness significantly. Higher temazepam concentrations produced drowsiness, reduced anxiety and impaired memory.

Residual effects after 24 hours

Table 4 shows the results of the digit symbol substitution, logical memory and mental control tests carried out immediately before, and 24 hours after, temazepam administration. No significant differences were found between the predose and 24-hour scores using the Wilcoxon matched-pairs signed-ranks test.

Discussion

Our clinical results confirm that temazepam is a useful drug in premedication with a rapid onset of action and sedative, anxiolytic and amnesic properties which persisted for between 2 and 5 hours. The mean peak plasma concentration (1560 ng/ml) and time to peak (49 minutes) observed in this study are in broad agreement with previously published data after adjusting for dose. Mean peak concentrations between 444 and 935 ng/ml after 20-mg doses[1,2,12] and 306 ng/ml after a 10-mg dose,[13] usually attained within one hour, have been reported previously. However, the relationship between temazepam concentration, clinical effect and duration of action has received scant attention in the past. Mattila *et al.*[2] demonstrated that the performance of volunteers in the digit symbol substitution test was impaired progressively with increase in temazepam concentration and it has been suggested recently that concentrations of 200–300 ng/ml are hypnotically active.[14] The results in Table 3 show clearly that concentrations in excess of 300 ng/ml produce demonstrable effects on memory, anxiety and wakefulness and that the intensity of these effects becomes more evident at higher concentrations. Absorption was rapid in all our patients, and despite some inter-individual variability effective concentrations were achieved within 30 minutes. Results from a pharmacokinetic study of temazepam elixir[12] suggest that an even more rapid clinical response could be achieved, if required, by using this formulation.

The biexponential decline in plasma concentration reported here confirms previous observations. The distribution half-life of 1.24 hours is consistent with values of 1.3 hours in young healthy volunteers[12] and 0.7 hours in geriatric patients.[13] In our study, plasma concentrations after the peak declined rapidly to subtherapeutic levels due to distri-

Table 4. Clinical assessments performed before and 24 hours after temazepam 40 mg mean (SD).

	Before administration	After 24 hours
Digit symbol substitution test	29.1 (10.2)	29.6 (9.4)
Mental control	5.9 (2.0)	6.1 (1.7)
Logical memory	7.8 (2.7)	7.8 (3.0)

bution into tissues, and the termination of the clinical effects coincided approximately with the end of the distribution phase (3–4 half-lives). A further slow decrease in temazepam concentration followed due to elimination but the elimination half-life is largely irrelevant to the duration of action when a single dose of temazepam is used for the purpose of premedication. The elimination half-life of 7.5 hours in this study is an underestimate because of insufficient blood sampling during the elimination phase; after single oral doses of temazepam in soft gelatin capsules, mean elimination half-lives of 9.8 hours[12] and 8.6 hours[15] have been reported in healthy volunteers, and 8.7 hours[13] in geriatric patients. Plasma temazepam concentrations were shown to be significantly higher in geriatric women than in men after daily doses of 10 mg for 2 weeks.[13] A study of the effects of age and gender on temazepam pharmacokinetics showed the elimination half-life to be prolonged in elderly women.[16] This could account for the relatively high concentration remaining after 24 hours in subject 5, although at this concentration no residual clinical effects were expected or observed. Our results showed no memory deficit or depression of psychomotor function at 24 hours; this is in agreement with sleep laboratory studies in which temazepam was found to be an effective hypnotic with no hangover effects.[17] This is partly due to the method of formulation; temazepam 30 mg in a hard gelatin capsule did reduce the choice reaction time and the critical flicker fusion test on the morning after its administration for insomnia.[18]

O'Boyle *et al.*[8] in contrast to our observations, found that patients aged between 18 and 32 years who underwent minor outpatient oral surgery were fully recovered 120 minutes after temazepam 40 mg. Psychomotor function had returned to baseline and the patients were judged to have 'street fitness'. Patients in our study were still drowsy 4 hours after they took this dose. The discrepancy between the results may be explained partly by the difference in age of the patients and partly by the different nature of the surgery.

In conclusion, our clinical results agree with those reported in adult volunteers[19] which showed a prolongation of clinical effects for more than 3.5 hours after oral temazepam 40 mg. We agree that temazepam cannot be considered to be an ultra-short acting hypnotic at this dose, but suggest that it may be appropriate in the anxious inpatient.

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Intravenous enalaprilat and autonomic reflexes

The effects of enalaprilat on the cardiovascular responses to postural changes and tracheal intubation

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Summary

Thirty healthy patients, who were to undergo surgery which required tracheal intubation, were given an intravenous injection of enalaprilat (either 0.5 mg, 1 mg, 2 mg or 4 mg; six patients for each dose) or normal saline 17 minutes before induction of anaesthesia with thiopentone 3–5 mg/kg, and suxamethonium 1.5 mg/kg. Postural manoeuvres were performed 5 minutes before and 6, 11 and 16 minutes after enalaprilat or saline. Complete inhibition of angiotensin converting enzyme occurred with all doses of enalaprilat, which allowed the four different treatment groups to be considered as one large treated group. The mean arterial pressure was almost unchanged during the postural manoeuvres; the heart rate increased, mostly similarly (by approximately 10%) in both groups. Mean arterial pressure in the recumbent position decreased over the 17 minutes before induction in the enalaprilat group, and increased slightly in the control group (treated mean, −5.0%; controls mean, 1.8%; difference, −6.8%; 95% confidence intervals of difference, −2.3 to −11.3%, $p < 0.01$). This difference was again seen after induction (treated, −8.0%; controls, 7.7%; confidence intervals of difference, −0.6 to −31%) and for a 5-minute period shortly after tracheal intubation. The increases in mean arterial pressure produced by intubation itself were similar in both groups (treated, +36%; controls, +35%; 95% confidence intervals of difference, −16% to +18%). Changes in heart rate after induction were also similar in both groups. It is concluded that intravenous enalaprilat acted as a hypotensive agent with a sparing effect on autonomic reflexes, both before and after induction of anaesthesia.

Key words

Pharmacology; angiotensin converting enzyme, enalaprilat.
Intubation, tracheal; complications.

Tracheal intubation during induction of anaesthesia usually causes transient hypertension and tachycardia.[1,2] These cardiovascular effects result from adrenergic hyperactivity and are usually not harmful in healthy individuals. However, in patients with hypertension, coronary artery and cerebrovascular disease such effects may not be harmless.[3-5] Vasodilators, narcotic analgesics, beta receptor blocking agents, local anaesthetics and various induction agents have all been used to prevent these responses with variable degrees of success.[6-15]

Angiotensin I is converted in the renin–angiotensin–aldosterone pathway to angiotensin II by the action of angiotensin converting enzyme (ACE). The actions of angiotensin II include: increased secretion of aldosterone from the adrenal cortex,[16] vasoconstriction due to direct action on vessel walls,[16] presynaptic facilitation of release of noradrenaline by a direct action on post-ganglionic neurons,[16] a vagolytic action,[17] and centrally mediated effects causing an increase in blood pressure.[18]

ACE inhibitors compete with angiotensin I for binding sites on the ACE molecule to inhibit the conversion of angiotensin I to angiotensin II.[19] If the production of angiotensin II is inhibited, its level falls rapidly as it is broken down by angiotensinases. The half life of angiotensin II is 1–2 minutes in humans.[20] Therefore, by reducing the effects of angiotensin II, ACE inhibitors cause a reduction in blood pressure.

It is suggested in addition that ACE inhibitors may have antihypertensive properties which are independent of the renin-angiotensin system. The mechanisms involved include the sympathetic nervous system, kinins, and prostaglandins.[21]

Enalaprilat is the active diacid of the ACE inhibitor enalapril maleate. It is inactive when given orally, but an intravenous formulation may be used in conditions when a rapid ACE inhibition response is required, for example, hypertensive emergencies and left ventricular failure.[22,23] This intravenous preparation is potentially useful in patients immediately before anaesthesia and surgery. We have therefore investigated the effects of intravenous enalaprilat on the cardiovascular responses to tracheal intubation. These responses result from hyperadrenergic activity,

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so the effect of enalaprilat on another autonomic reflex, that caused by a rapid change in posture, was also examined.

Method

The study was approved by the hospital Ethics Committee and written informed consent was obtained from each participating patient.

The study was performed on patients aged between 18 and 65 years, ASA grade 1 or 2, who weighed 50–90 kg, and were to have surgery which required the trachea to be intubated. Patients who were hypertensive, or on medication which influenced blood pressure, and those in whom pregnancy was a possibility were excluded.

Twelve patients in the first part of an open study were randomly allocated to two groups, a control group and those treated with 1 mg intravenous enalaprilat; later, 18 patients were randomly allocated to three further groups who received either 0.5 mg, 2.0 mg or 4.0 mg intravenous enalaprilat.

All patients were premedicated with 10 mg oral diazepam 1–2 hours before operation. Each patient was connected in the anaesthetic room to an ECG monitor and an intravenous cannula inserted. Arterial blood pressure (systolic, diastolic and mean) and heart rate were measured noninvasively in the opposing arm using a Critikon Vital Signs Monitor (Dinamap™ model no. 1846). A Critikon printer (model TR2000) was used to record the data. Measurements were made at 1-minute intervals after the first reading until 10 minutes after induction of anaesthesia. Additional measurements were made at 30-second intervals during the postural manoeuvres which were carried out 5 minutes before, and 6, 11 and 16 minutes after the injection of enalaprilat or saline. The postural change was accomplished by a rapid tilt of the operating table with a foot support attached, to a head-up position at approximately 60° to the horizontal.

The blood pressure and heart rate were monitored for 10 minutes before an intravenous injection of enalaprilat or saline. The readings during this first 10-minute period were used to calculate each patient's average pretreatment blood pressure and heart rate; those measured during and immediately after the postural change were omitted. The readings at 17 minutes were used as the pre-induction blood pressure and heart rate.

Blood samples were taken immediately before enalaprilat or saline was given, and thereafter at 1, 5, 10, 20, 30 and 60 minutes. The samples were subsequently analysed in order to measure renin activity and to monitor both ACE activity and enalaprilat levels during the first hour after the administration of the drug (Appendix A).

Anaesthesia was induced one minute after the final tilt (17 minutes after the injection of enalaprilat) with thiopentone 3–5 mg/kg, injected at a rate of 100 mg per 10 seconds until the eyelash reflex was lost, followed by suxamethonium 1.5 mg/kg. Ventilation of the lungs was controlled with 50% nitrous oxide in oxygen, until laryngoscopy and tracheal intubation were performed 90 seconds after the injection of the muscle relaxant. The concentration of nitrous oxide was increased after intubation to 67%, and 1% enflurane was introduced until the blood pressure had returned to the pretreatment level. The volatile agent was subsequently adjusted to maintain the blood pressure at this level. Atracurium 0.5 mg/kg was given 5 minutes after induction, or sooner if signs of neuromuscular recovery were observed. All inductions and intubations were performed by the same investigator, and all patients were monitored in the recovery area and, later, in the ward.

Statistical analysis was performed in terms of mean arterial pressure (MAP) and heart rate, and involved the use of 95% confidence intervals (CI), unpaired *t*-tests, and one way analysis of variance as appropriate. Significant differences were assumed present when $p < 0.05$.

Results

Differences of mean values of age, weight, MAP, heart rate and of the female/male ratio between the five groups were small (Table 1). Mean plasma renin activity was higher in the control group than the treated groups because one normotensive control patient had an unusually high renin activity (12.3 ng/ml/hour). The predominance of females in all groups was due to a large number of gynaecological patients.

Mean plasma levels of enalaprilat decayed roughly exponentially with time and, at any measurement, were roughly proportional to dose as seen in Fig. 1 (note the logarithmic *y*-axis). ACE activity was not detectable (< 1 IU/litre) in 134 out of 144 blood samples, from the 24 patients who had received various doses of enalaprilat. The ACE activities in the other 10 samples were very low and are shown in Table 2. These unexpected results were used as justification for the amalgamation of the four groups with different doses of enalaprilat into a single treated group of 24 patients to be compared with the control group of six patients.

The blood pressure of a patient who had received 1.0 mg of enalaprilat decreased to 47/29 (MAP 40) mmHg after induction of anaesthesia from a pretreatment level of 124/71 (95) mmHg and a pre-induction level of 118/75 (90) mmHg. Her trachea was intubated immediately, one minute before the scheduled time, and her blood pressure increased to 147/91 (113) mmHg. She remained normotensive for the rest of the anaesthetic and showed no ill-effects

Table 1. Demographic data. Values expressed as mean (SD or range).

Dose of enalaprilat, mg	0.0 $n = 6$	0.5 $n = 6$	1.0 $n = 6$	2.0 $n = 6$	4.0 $n = 6$
Sex, female/male	6/0	5/1	6/0	4/2	5/1
Age, years	37 (29–47)	35 (29–39)	42 (28–62)	40 (35–50)	37 (31–51)
Weight, kg	69 (12.0)	64 (4.2)	64 (6.6)	66 (6.0)	68 (8.5)
Mean arterial pressure, mmHg	87 (7.7)	92 (11.3)	91 (7.2)	85 (11.4)	91 (8.2)
Heart rate, beats/minute	75 (17.0)	71 (13.0)	79 (13.8)	65 (11.0)	73 (13.2)
Renin activity, ng/ml/hour	3.5 (0.8–12.3)	2.3 (0.6–3.8)	2.6 (1.7–4.8)	2.1 (1.4–3.0)	3.0 (1.4–5.9)

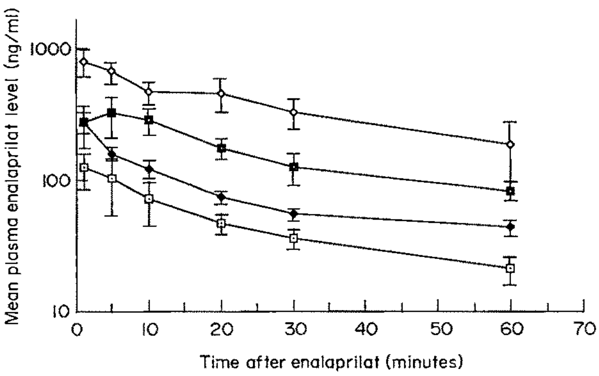

Fig. 1. Plasma enalaprilat levels expressed as mean (SEM) for patients who received 0.5 mg enalaprilat, ⊡; 1.0 mg enalaprilat, ◆; 2.0 mg enalaprilat, ■; or 4.0 mg enalaprilat, ◇. Plasma levels varied significantly with dose at all times ($p < 0.01$).

Table 2. ACE activities (IU/litre) which were detectable in individual treated patients after enalaprilat (numbers 1–6) and mean values (SD) of ACE activity for the control and treated groups.

	Interval after enalaprilat (minutes)							
Patient	0	1	5	10	20	30	60	dose (mg)
1	395	0	0	0	0	6	12	0.5
2	330	0	0	0	0	0	6	0.5
3	215	0	0	0	0	0	24	0·5
4	306	0	0	2	0	0	0	1
5	356	0	13	0	0	2	40	1
6	367	19	0	0	13	0	0	1
Controls	344	325	320	340	301	312	301	nil
$n = 6$	(139)	(118)	(128)	(130)	(118)	(137)	(113)	
Treated	316	1	1	0	1	0	3	0.5–4
$n = 24$	(114)	(4)	(3)	(0)	(3)	(1)	(10)	

afterwards. However, her cardiovascular measurements were excluded from analysis at all times after this hypotensive episode which had caused a breach of the protocol.

Mean changes of MAP from pretreatment levels for the treated group and the control group are shown in Fig. 2. Statistical analyses of these changes are shown in Table 3. MAP decreased in the treated group after enalaprilat, so that after 15 minutes there was a significant difference in mean change of MAP between the groups. After induction, but before intubation, the results were more variable (see the error bars in Fig. 2), but the difference was larger and still significant at 18 minutes. Intubation produced the expected increase in MAP in both groups. The difference between the groups persisted for another 7 minutes and was significant from 2 to 6 minutes after intubation.

The highest mean MAP in both groups was measured at one minute after tracheal intubation. Compared to the pretreatment MAP, the mean change (due to enalaprilat, induction and intubation) was substantially, but not significantly, less in the treated group: treated, 32.2%; controls, 46.3%; difference, −14.1%; 95% CI of difference, −30.5 to +2.3% $p > 0.05$. However, compared to the after induction, before intubation MAP, the mean changes (due solely to intubation) were almost the same in both groups: treated, 35.7%; controls, 34.7%; difference, 1.0%; 95% CI of difference, −15.8% to +17.8%, $p > 0.05$.

Mean changes of MAP from pretreatment levels were calculated at each measurement time for each group. Analysis of variance of the four treated groups showed no significant difference between doses at any of the times. This was considered further justification for combination of the treated groups.

Mean heart rates for the control groups and the treated group throughout the study period are shown in Fig. 3. There were no significant differences in heart rate, or in the change of heart rate from pretreatment or before induction rates, between the control and treated groups at any time.

The responses to the postural manoeuvres are shown in Table 4. The mean MAP changes were very small and not significantly different between treated and control groups. There was a formally significant difference between the groups in the change of heart rate from pretilt level to that during the tilt at 11 minutes after enalaprilat. However, since this was only just significant, and only one of four identical comparisons (at different times), it can reasonably be attributed to chance. Heart rate changes were similar in

Table 3. Statistical analysis of Figure 2.

Time after enalaprilat (minutes)	Treated mean ($n = 24$)† (%)	Control mean ($n = 6$) (%)	Difference between means (%)	95% CI (%)	Significance
5	−3.6	−1.0	−2.6	−10.1 to +4.9	
10	−6.2	−0.5	−5.7	−12.6 to +1.2	
15	−5.0	1.8	−6.8	−11.3 to −2.3	**
17	−5.2	0.7	−5.9	−12.8 to +1.0	
Induction					
18	−8.0	7.7	−15.7	−30.8 to −0.6	*
19†	−1.4	8.4	−9.8	−22.1 to +2.5	
Intubation					
20	32.2	46.3	−14.1	−30.5 to +2.3	
21	19.2	38.0	−18.8	−30.5 to −7.1	**
22	4.8	20.7	−15.9	−27.3 to −4.4	**
23	−1.8	12.9	−14.7	−25.6 to −3.8	*
24	−6.6	7.9	−14.5	−27.2 to −1.8	*
25	−8.5	9.8	−18.3	−32.2 to −4.4	*
26	−9.2	−2.1	−7.1	−19.8 to +5.6	
27	−9.4	−11.3	1.9	−12.7 to +16.5	

*$p < 0.05$; **$p < 0.01$.
†$n = 23$ for treated group from 19 minutes.

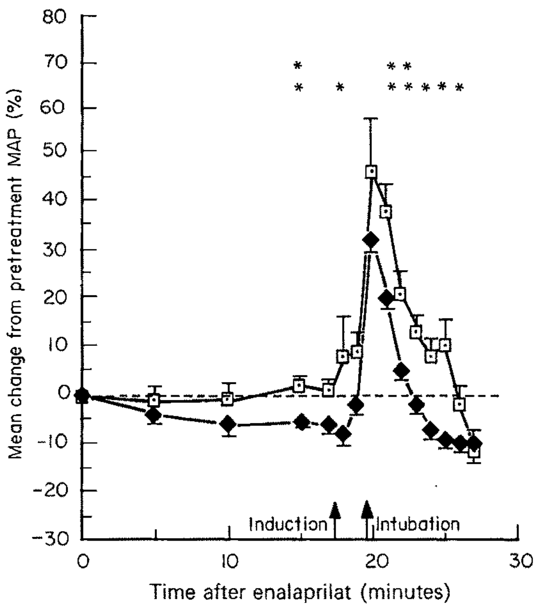

Fig. 2. Changes from pretreatment MAP expressed as mean (SEM) for controls, ⊡; and treated patients, ◆. Significant differences indicated by * = $p < 0.05$, and ** = $p < 0.01$.

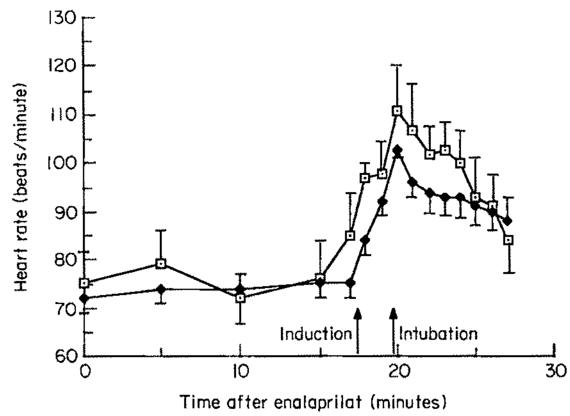

Fig. 3. Heart rate expressed as mean (SEM) for controls, ⊡; and treated patients, ◆. There were no significant differences at any time between the groups.

the two groups during the postural manoeuvres before enalaprilat, and at 6 and 16 minutes after enalaprilat.

No adverse effects were observed after the study period.

Discussion

Orally administered ACE inhibitors have an established place in the management of hypertension[24] and heart failure.[25] In addition, captopril has been shown to reduce the need for sodium nitroprusside (SNP) in SNP-induced hypotension for major orthopaedic surgery.[26] More recently it has been suggested that enalapril is associated with reduced pressor responses to tracheal intubation and surgical stimulation.[27] However, orally administered drugs have the disadvantages of variable absorption, onset and duration of action.

The introduction of intravenous preparations of ACE inhibitors may result in increased use of these drugs in anaesthesia. This study was designed to investigate the use of a new intravenous ACE inhibitor, enalaprilat, and was open in order to gain experience in acute ACE inhibition with anaesthesia and surgery. The objective data generated by the study made its open nature less disadvantageous.

Our results showed that intravenous enalaprilat caused a significant reduction in MAP before induction of anaesthesia and before intubation (Fig. 2). The maximum increase in MAP from the pretreatment level was also reduced to a similar extent, but it cannot reasonably be suggested that enalaprilat reduced the *pressor response to intubation*. Furthermore, the failure to demonstrate any difference in the autonomic response to a postural change, leads us to suggest that enalaprilat acted as a hypotensive agent with a sparing effect on autonomic reflexes; this may be a useful property since it raises the possibility that any hypotension associated with enalaprilat might be reversed by sympathomimetic drugs.

Maximum ACE inhibition was seen with all doses of enalaprilat used in the study (Table 2). ACE activity only started to return in the groups who had the lower doses (0.5 mg and 1.0 mg) after 60 minutes. These results may be a little biased because of *in-vitro* binding of enalaprilat with ACE after the blood samples were drawn from the patients. However, the rapidity of the development of a hypotensive effect coupled with the knowledge of the natural half-life of angiotensin II (1–2 minutes),[20] suggests that ACE inhibition was present at the times when the samples were taken.

Table 4. Mean values (SEM) of changes in mean arterial pressure and heart rate at each postural manoeuvre, with mean differences and 95% confidence intervals.

	Before enalaprilat	Time after enalaprilat (minutes) 6	11	16
Mean arterial pressure				
Treated, $n = 24$ (%)	0.2 (2.0)	−1.8 (1.8)	−0.4 (1.8)	−1.5 (1.4)
Controls, $n = 6$ (%)	−3.8 (2.3)	1.2 (2.3)	−6.2 (3.6)	−0.8 (3.0)
Mean difference (%)	4.0	−3.0	5.8	−0.7
95% CI (%)	−4.6 to +12.6	−10.8 to +4.8	−2.4 to +14.0	−7.3 to +5.9
Heart rate				
Treated, $n = 24$ (%)	9.8 (2.1)	9.3 (2.6)	9.8 (1.7)	9.7 (2.4)
Controls, $n = 6$ (%)	10.7 (5.2)	10.2 (5.0)	20.2 (5.9)	11.7 (6.3)
Mean difference (%)	−0.9	−0.9	−10.4	−2.0
95% CI (%)	−10.9 to +9.1	−12.8 to +11.0	−19.4 to −1.4*	−13.7 to +9.7

*$p < 0.05$.

The ACE activity results also suggest that the lowest dose used in this study (0.5 mg) may be sufficient for an effective response (i.e. complete ACE inhibition) of the shortest duration, in the order of one hour. Despite the high drug levels and therefore presumably prolonged ACE inhibition in patients who had the larger doses, hypotension was not observed in the recovery ward. This may be because intravenous fluids were used liberally in patients who were considered hypotensive for any reason, after the end of the study period, which was 10 minutes after induction.

ACE inhibitors are, effectively, vasodilators[28] but their use is unaccompanied by a reflex tachycardia. This was confirmed in the present study and could be advantageous when an increase in heart rate may be deleterious. The likely mechanisms for the absence of a reflex tachycardia with ACE inhibitors include a central sympatho-inhibitory action, removal of peripheral angiotensin-II mediated presynaptic facilitation of noradrenaline release, unchanged arterial wall tangential tension during ACE inhibition, selective venodilatation, parasympathetic activation and resetting of the baroreceptor-mediated reflexes.[17,29–33]

One patient, a fit, 62-year-old, 69-kg woman, developed an unacceptable degree of hypotension, albeit of short duration. This may have been the result of enalaprilat (1 mg), thiopentone (250 mg) or more likely, the interaction between these two drugs. If this unacceptable hypotension was caused by enalaprilat, it demonstrates the reversible nature of such hypotension, corrected in this case by the patient's pressor response to intubation. Nevertheless, it should be noted that there is a possibility of profound hypotension when enalaprilat is used in combination with anaesthetic agents, even in fit patients. Patients with severe heart failure or volume-depletion (e.g. from diuretic therapy or dialysis) are more likely to develop severe hypotension with the first dose of oral ACE inhibitors,[34] and this could be an even greater problem with an intravenous preparation.

We believe, in conclusion, that intravenous ACE inhibitors, of which enalaprilat is an example, may have a useful place in anaesthesia as rapidly acting, hypotensive drugs which have minimal effects on autonomic reflexes. We therefore suggest that it is appropriate to investigate intravenous enalaprilat further in controlled clinical trials.

Appendix A

Plasma renin activity was measured as angiotensin I generating activity of plasma in ng/ml/hour. Angiotensin I was measured by radioimmunoassay. The sensitivity of this method was 0.1 ng/ml/hour. ACE activity was measured by a colorimetric assay of glycyl-glycine liberation from hippuryl-glycyl-glycine, in IU/litre with a sensitivity of 1 IU/litre. Serum enalaprilat concentrations were measured by double-antibody radioimmunoassay with a sensitivity of 0.4 ng/ml. All analyses were performed at the Institute of Biopharmaceutics, Monksland, Athlone, Ireland.

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The effects of secondary transport on critically ill patients

S. RIDLEY AND R. CARTER

Summary

This study examined the effect of secondary transport on critically ill patients and the effectiveness of a regionally based intensive care service. Four hundred and ninety-five patients were studied retrospectively over a 2-year period. Eighty-two were transferred from peripheral hospitals in a mobile intensive care unit while the remaining 413 were admitted directly to the intensive therapy unit at the Western Infirmary, Glasgow. The severity of illness in both groups was assessed using the APACHE II scoring system. The transferred group were scored before and after the journey, while the directly admitted group were scored only on admission. The results show that the transferred patients exhibited a consistent cardiorespiratory response to transport irrespective of their severity of illness, and that the mortality in both groups of patients in the intensive therapy unit was not significantly different. The results also suggest that in the transferred group, the outcome is not only dependent on the severity of illness but also on other factors, such as the hospital from which the patient was referred and the duration of the pretransfer admission.

Key words

Critical care; transport.

Secondary transport involves the transfer of critically ill patients from one hospital to another centre which is staffed and equipped more appropriately for the patient's further management. These patients are transferred most appropriately in a specialised mobile intensive care unit (MICU) and their care is usually supervised by medically qualified personnel.

A working party of the Association of Anaesthetists of Great Britain and Ireland examined the provision of intensive care recently[1] and recommended that expensive intensive care resources should be concentrated in regional centres. Implementation of this recommendation will increase the future requirement for secondary transfer. However, Wright *et al.*[2] found the facilities for secondary transfer deficient in the UK as a whole. Sixty-five percent of intensive therapy units (ITU) had no dedicated vehicle for MICU use and just under 80% had no modified trolley on which to base a MICU. More MICUs will need to be established and experience gained in the safe transfer of critically ill patients if rationalisation of intensive care services is to proceed.

Several studies have revealed that, without care and supervision, patients frequently suffer further untoward insults during the journey.[3–5] However, seriously ill patients may be moved without deterioration if thorough assessment and stabilisation are undertaken before transfer, and continuous and close monitoring employed during the journey.[6]

The aim of this study was to examine the effects of secondary transfer on critically ill patients and to identify factors, other than the severity of illness, which might contribute to the patients' eventual outcome.

Methods

The study examined retrospectively all admissions to the ITU at the Western Infirmary, Glasgow, over a 2-year period (July 1985 to June 1987). Four hundred and ninety-five patients were studied; 413 were admitted directly to the ITU from other wards and departments in the hospital and 82 were transferred in the MICU from peripheral hospitals.

Each patient's severity of illness was assessed using the APACHE II scoring system developed by Knaus *et al.*[7] However, because haemoglobin concentration rather than haematocrit is measured more commonly in this country, the ranges of haemoglobin values proposed by Farman[8] were used instead.

The patients admitted directly to the ITU (control group) were scored once on admission. Patients who were transferred were scored on four occasions: at the onset of the critical illness which precipitated referral to the Western Infirmary (APACHE score 1); before departure from the peripheral hospital (APACHE score 2); on arrival at the ITU at the Western Infirmary (APACHE score 3); and after 24 hours of intensive care management (APACHE score 4). The demographic details of all patients and the

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Table 1. Demographic details.

	Direct admissions	Transferred patients
Age, years		
Mean	53	44
SD	18	18
Range	16–88	17–79
Male : Female ratio	1.2 : 1	1.3 : 1

clinical conditions from which they were suffering were recorded; in the transferred group, details of the reason for referral and duration of admission at the peripheral hospital were also noted.

All data were extracted from the case notes. The data were stored on a computer database and the APACHE scores were calculated using a programme checked by multiple manual calculations of the score.

The statistical methods used included the Wilcoxon Rank Sum and Kruskal–Wallis tests for analysis of the APACHE scores, and the Chi-square and Student's *t*-tests were performed when frequency or normally distributed parametric data were analysed. Stepwise logistic regression was employed to assess the effect of increasing pretransfer admission time and APACHE score on outcome in the transfer group. A p-value of less than 0.05 was considered significant. Isotonic regression (pooled adjacent violators algorithm)[9] was used to calculate the expected mortality of patients at the Western Infirmary ITU for each APACHE score (range 0 to > 30). This method was chosen because of the previously demonstrated nonlinear nature of increasing mortality with increasing APACHE score.[7] The control group was subdivided into an earlier two-thirds and a later one-third. In the earlier subgroup, the percentage of nonsurvivors was calculated for each APACHE score value. Using this percentage as the expected probability of death, the number of deaths in each APACHE score group was predicted in the later one-third of the control patients. The predicted number of deaths and the actual number of deaths in this later group were not significantly different. After validation of the expected probability of mortality in the two subgroups, the expected mortality for each APACHE score was recalculated for the control group as a whole.

Results

Sufficient data were retrieved in the directly admitted group to allow calculation of an accurate APACHE score in all but seven of 413 patients. In the transferred group, data retrieval allowed scoring at the onset of the critical illness (APACHE Score 1) in 66 patients, and 60 patients were scored before departure from the peripheral hospital (APACHE Score 2). Data for two patients were not available on admission to the ITU and this prevented calculation of APACHE score 3. Seventy-one patients were scored satisfactorily after 24 hours on the ITU (APACHE Score 4).

The demographic details of patients in both groups are shown in Table 1. The clinical categories which warranted admission to ITU together with their respective ITU mortality are shown in Table 2. The primary reason for direct admission to the ITU from other wards and departments at the Western Infirmary was respiratory support (33%), while the predominant reasons for transfer were management of extensive trauma (20%) or cardiovascular problems (18%). The types of unit from which the patients were transferred and the principal reason for referral are shown in Table 3. For 65% of patients, the principal administrative reason for referral to the Western Infirmary was the

Table 2. The clinical categories (and respective percentage mortality) of all the patients studied.

	Patient numbers (and percentage mortality)	
	Control group	Transfer group
Neurological	9 (11)	5 (20)
Cardiovascular		
Haemorrhagic shock (including abdominal aneurysm)	35 (46)	1 (0)
Septic shock	25 (68)	13 (8)
Severe bleeding	11 (18)	0
Others	8 (50)	1 (0)
Gastrointestinal		
Obstruction/perforation	38 (13)	14 (28)
Respiratory		
Failure/postoperative IPPV	86 (14)	1 (0)
Infection	49 (14)	9 (22)
Trauma	53 (13)	16 (6)
Poisoning		
Self	25 (8)	5 (60)
Toxic	5 (40)	1 (100)
Postcardiac arrest	21 (52)	0
Two-system failure (including renal failure)	35 (14)	11 (72)
Miscellaneous	13 (31)	5 (40)

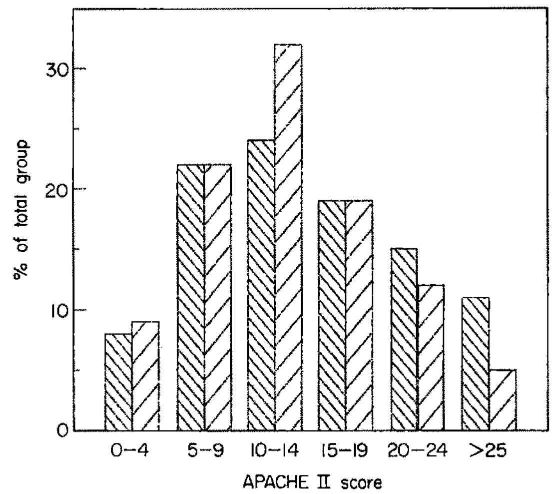

Fig. 1. Distribution of APACHE II scores in patients admitted directly ▧; and those who were transferred from other hospitals ▨.

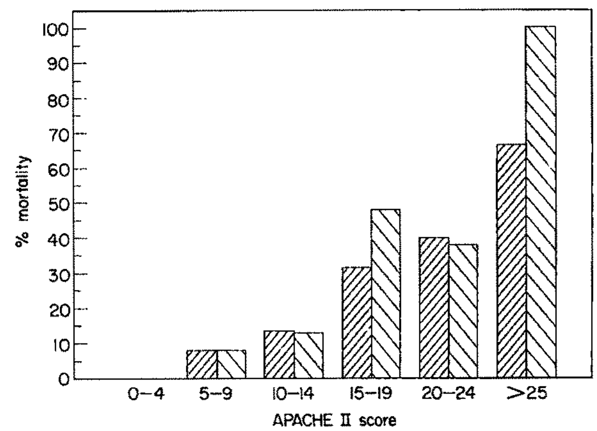

Fig. 2. Percentage mortality related to APACHE II score. ▨, patients admitted directly; ▧, patients transferred from other hospitals.

lack of facilities for long-term ventilation of critically ill patients.

Figure 1 illustrates the range of severity of illness as assessed by the APACHE score; the percentage mortality for each APACHE score group is illustrated in Figure 2. These histograms show that the distribution of APACHE scores is similar in the control and transferred patients. There was no significant difference between the ITU mortality of the two groups.

The APACHE score before departure from the peripheral hospital (APACHE Score 2) was compared with the APACHE score on arrival at the Western Infirmary ITU (APACHE Score 3) to assess the effect of secondary transport on the patients. These two scores were calculated satisfactorily in 58 of the transferred patients. These patients were classified into three groups according to APACHE score before departure (< 10, 10–19 or ≥ 20). Table 4 shows the changes in APACHE score which occurred during transfer. The mean score in patients with an APACHE score of less than 10 increased on arrival at ITU while the mean score in patients with the highest initial scores ($\geqslant 20$) was lower on arrival at the Western Infirmary ($p < 0.05$). The intermediate APACHE score group demonstrated no significant change in score during transfer.

Examination of the physiological parameters which contribute to the final APACHE score revealed six which were most likely to change (Table 5). There were statistically significant changes in mean arterial pressure, heart rate, oxygenation (as measured by the alveolar–arterial

Table 3. The type of unit and the reasons for referral (frequently, patients were referred for more than one reason).

	Peripheral hospital details	
Type of unit	Number of hospitals	Numbers of patients transferred
Intensive care	10	29
High dependency	2	23
No capacity for ventilation	10	30

Reasons for referral

Reason	Incidence (%)
No capacity for long-term ventilation	54
No dialysis facilities available	20
Further ITU management required	17
No acute medical services	3
Referral to cardiothoracic unit required	2
Inappropriate ITU (usually from neurosurgical centre)	4

Table 4. The changes in APACHE scores from baseline after transfer (* = $p < 0.05$).

APACHE score	*n*	Mean change (SD)	Range	Centile 10th	Centile 90th	
< 10	15	+2.1 (2.9)	+8 – −2	+7	−1	*
10 < score < 19	34	+0.5 (4.2)	+11 – −9	+6	−5	
⩾ 20	9	−2.8 (2.6)	0 – −6			*

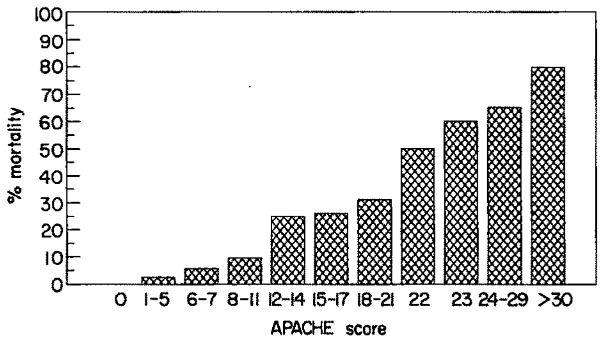

Fig. 3. Expected mortality of patients admitted directly to the ITU (data from 408 patients in whom the APACHE II score was calculated satisfactorily).

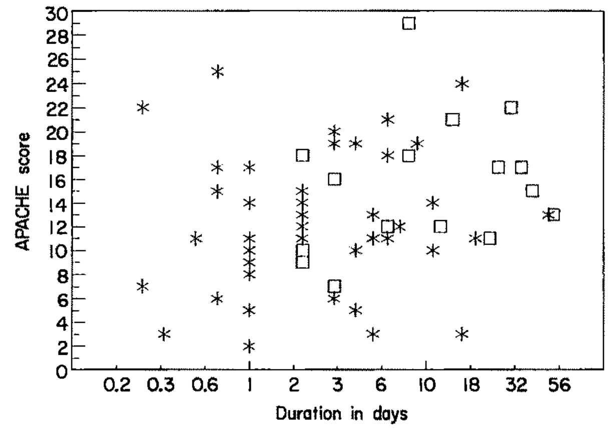

Fig. 4. Duration of admission to hospital before transfer in relation to APACHE II score and mortality after transfer. ✱, survivors; □, non-survivors.

oxygen tension difference) and haemoglobin concentration. The direction of changes in these variables was the same in each of the three subgroups (i.e. APACHE score < 10, 10–19, $\geqslant 20$).

Over the 2-year period, five hospitals referred more than five patients each to the Western Infirmary. A total of 57 patients were referred from these hospitals and 49 of these were scored satisfactorily before departure. Table 6 shows the APACHE II scores and numbers of deaths in patients referred from the five peripheral hospitals. The expected number of deaths in these patients was derived from their APACHE II score and the expected probability of mortality of the control group (Fig. 3), with the assumption that the treatment and care provided in the MICU and the ITU at the Western Infirmary was applied uniformly and consistently. The actual number of deaths divided by the expected deaths in this group is expressed as a mortality ratio for each hospital. However, the differences do not reach statistical significance because of the small numbers involved.

Statistical analysis revealed that any suspected variation in mortality ratios was not related to the severity of illness of the patients. Hospital C has a mortality ratio of 0.29 but referred more critically ill patients as judged by the significantly higher APACHE scores ($p < 0.05$).

The actual and predicted mortality for the 60 transferred patients in whom the APACHE II score was obtained before transfer is shown in Table 7.

Figure 4 illustrates the effects of high APACHE II score and the duration of pretransfer admission on the ITU outcome in the transferred group. As expected, higher APACHE scores increased the risk of death significantly ($p < 0.02$) but the effect of increasing duration of pretransfer admission failed to reach significance ($p = 0.066$).

Discussion

Eighty-seven percent of the scores were completed satisfactorily in the transferred group. However, this is an underestimate of the success of data retrieval because some patients were referred to the Western Infirmary within a few hours of the onset of critical illness and therefore APACHE scores 1 and 2 coincided. In addition, some patients died within 24 hours of arrival at the Western Infirmary. The APACHE score was not calculated if more than two of the variables which make up the score were not recorded in the notes. If only one or two variables were not recorded, then the missing value(s) were either assumed to be normal or estimated from measurements recorded around the same time. The case sheets of seven patients in the control group were untraceable and this prevented scoring.

The age ranges, sex ratio and distribution of severity of illness were similar in both the control and transferred groups, but the clinical categories differ. This may reflect the capabilities of the peripheral hospitals, in that respiratory failure from whatever cause can be managed satisfactorily in the short term but when other problems such as cardiovascular derangement, trauma, intestinal obstruction and perforation compound the respiratory failure, transfer is requested. Chang *et al.*[10] have reported that mortality depends not only on the actual APACHE score but also on how long the patients have had a particular score. The transferred patients had spent longer in hospital after the onset of their critical illness than the control patients, but these patients had a similar ITU mortality irrespective of their severity of illness. This may be a reflection of the

Table 5. The physiological parameters which changed during transfer (* = $p < 0.05$). Means, standard deviations and (range) given.

	Before transfer	Changes after transfer	
Temperature (°C)	37.1, 1.0 (36.4–38)	−0.45, 1.5 (−5.9–+2.8)	
Mean arterial pressure (mmHg)	82, 20 (23–127)	+10, 22 (−29–+77)	*
Heart rate (beats/minute)	107, 21 (60–180)	−8, 19 (−60–+29)	*
Oxygenation $D(A\text{-}a)O_2$ (mmHg)	226, 171 (14–647)	+49, 147 (−301–+428)	*
pH	7.37, 0.12 (7.03–7.6)	+0.02, 0.01 (−0.17–+0.42)	
Haemoglobin (g/dlitre)	11.0, 2.3 (6.7–16.4)	−0.5, 1.9 (−7.1–+3.9)	*

Table 6. APACHE II scores before departure from the peripheral hospital and the mortality ratios for patients from each hospital.

	Hospital				
	A	B	C	D	E
	2	5	3	1	17
	8	6	5	3	17
	9	7	6	7	19
	11	9	12	10	
	12	9	14	11	
	12	11	15	11	
	12	11	17	14	
	13	13	19	15	
	13	18	20	16	
	14	18	21		
	22	18	25		
	24	19	29		
		21			
Actual deaths	3	5	1	2	2
Predicted deaths	2.90	2.30	3.40	1.20	0.80
Mortality ratio	1.03	2.20	0.29	1.66	2.41

different clinical categories (and hence potential mortality) in the transferred group. For example, no patient with severe bleeding, haemorrhagic shock, or who had suffered cardiorespiratory arrest was transferred. This implies that these patients either recovered or died in the peripheral hospital. Such inadvertent selection tends to bias favourably the referral of those patients with conditions which had a better outlook, and so improve the mortality figures for the transfer group as a whole. However, it is possible also that patients who had been seriously ill but who recovered rapidly were not referred to the Western Infirmary, and this may have resulted in an opposing bias.

Bion *et al.*[5] noted that patients display a response to movement during transport but they were unable to identify it precisely. The results of the present study show that patients responded by an increase in mean arterial pressure, a decrease in heart rate and haemoglobin concentration and a marked decrease in adequacy of oxygenation. It is difficult to explain these changes on the basis of a single mechanism. Waddell *et al.*[11] found that simulated ambulance transport increased arterial pressure and cardiac output in shocked dogs. Hothersall *et al.*[12] reported both hypotension and hypertension after ambulance transport, and transient hypertension and arrhythmias associated with sudden acceleration forces during the journey. They found that the lasting effects were minimised if the patients were resuscitated adequately before transport and sedated effectively during movement. It has been shown that vibration may cause either hypertension or cardiovascular depression, depending on the dominant frequency.[13] The decreases in haemoglobin concentration and heart rate in our patients may have been a response to the intravenous fluids given during pretransfer stabilisation. Generally, at least 500 ml of fluid was administered to offset the hypotension which would otherwise occur when the patient is moved to and from the MICU trolley. All patients are sedated during transfer, but the increase in arterial pressure may have been caused by an incompletely obtunded noradrenergic stress response.

The increase in alveolar–arterial oxygen tension difference may be in part an artifact. Usually, the patient's inspired oxygen tension was adjusted to give a reasonable safety margin during transfer. Benatar *et al.*[14] have reported that if the pulmonary shunt fraction is greater than 25%, as is likely in these critically ill patients, an increase in inspired oxygen tension does not result in a parallel increase in arterial oxygen tension. In these circumstances, an increased inspired oxygen fraction increases the alveolar–arterial oxygen tension gradient and hence the APACHE score. Unfortunately the mixed venous oxygen tension is not measured routinely and so precise conclusions about the degree of change in oxygenation cannot be made.

The response to transfer in patients with a good prognosis (i.e. APACHE score < 10) did cause a statistically significant increase in APACHE score, although this may not be matched by a clinical deterioration. However, in the patients with a poor prognosis (i.e. APACHE score $\geqslant 20$), movement may have caused the patient's physiological variables to move into a less abnormal range and thus reduced the APACHE score. Intensive care management is often instigated at the peripheral hospital, and by the time the patient arrives at the Western Infirmary, clear improvement in the patient's condition (and hence APACHE score) is seen. It is important to appreciate that these changes do occur during transport, so that the related problems may be anticipated during the pretransfer stabilisation.

Knaus *et al.*[15] demonstrated in the USA that ITU mortality in different hospitals varied, and was dependent upon factors which were unrelated to the patient's severity of illness. The number of patients transferred from hospitals A–E in the present study are small, but these results may indicate that the situation is similar in this part of Great Britain.

The duration of admission in the peripheral hospital before transfer may affect outcome. Only one of seven patients treated in the peripheral hospital for more than 19 days survived to leave the ITU at the Western Infirmary.

The risks of secondary transport measured in terms of mortality *en route* are small. All of these patients are critically ill, and their medical condition poses a greater

Table 7. The predicted deaths in the transfer group in relation to the APACHE II score before transfer.

APACHE II score	*n*	Expected deaths	Actual deaths
0–5	7	0.18	0
6–7	5	0.28	1
8–11	15	1.43	3
12–14	12	3.00	2
15–17	8	2.08	4
18–21	8	2.49	3
22	2	1.11	1
24–29	3	1.96	1
Totals	60	12.50	15

threat to their life expectancy than the actual process of movement. The study demonstrates that the risk of mortality *en route* is less than 1 in 82, although we suspect that it is considerably lower; the specialist secondary transport team in Glasgow has moved approximately 2500 critically ill patients over the last 12 years without mortality during transfer.

The effectiveness of secondary transfer and subsequent ITU progress is difficult to establish in a strictly scientific manner because this would require a randomised trial in which patients who had been referred to a regional intensive care unit were allocated either to remain for further treatment at the peripheral hospital or to be transferred by the MICU. At present this is unacceptable medical practice and the best that can be achieved is an observational study such as this.

In conclusion, patients may be transferred between hospitals safely while in a critical state because their eventual outcome is more likely to be determined by other factors. Irrespective of their severity of illness, patients exhibit a cardiorespiratory response to transport which may result from the therapeutic interventions performed before transfer and the stress of the transfer itself. Awareness of this response is important for safe transfer. The mortality of the transferred patients may be influenced by the duration of the pretransfer admission and possibly by the management in the hospital from which they were transferred.

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CASE REPORT

'Cured' myasthenia gravis and neuromuscular blockade

A. B. LUMB AND I. CALDER

Summary

A symptomless myasthenic patient who played representative squash received 0.1 mg/kg of vecuronium and enflurane as part of a general anaesthetic for elective gynaecological surgery. Neuromuscular block was prolonged. The effect of neuromuscular blocking agents and volatile anaesthetics in symptomless myasthenics is discussed. We conclude that these patients should be assumed to be sensitive to such agents.

Key words

Anaesthetics, volatile; enflurane.
Neuromuscular relaxants; vecuronium.

An abnormal sensitivity to non-depolarising muscle relaxants is a feature of myasthenia gravis, but there are few reports about the response of myasthenic patients in remission. We describe the case of a symptomless myasthenic patient who responded abnormally to neuromuscular blockade.

Case history

A 30-year-old Caucasian woman, who weighed 71 kg, presented for an elective laparoscopy and minilaparotomy as part of investigation into her secondary infertility. Myasthenia gravis was diagnosed 12 years previously, and treated initially with pyridostigmine, azathioprine, and steroids, followed one year later by a thymectomy, which rendered her asymptomatic with no treatment. For the 9 years before this admission her exercise tolerance had been excellent; she enjoyed playing squash and regularly represented her club. She received general anaesthesia for a laparoscopy 3 years previously, but neuromuscular blocking agents were not used. The patient was fit and well on admission, with no signs of myasthenia, and she was taking no medication.

She was premedicated with papaveretum 10 mg and hyoscine 0.2 mg intramuscularly 1.5 hours before operation. Anaesthesia was induced with thiopentone 250 mg, and 7 mg (0.1 mg/kg) vecuronium to produce neuromuscular blockade. Intermittent positive pressure ventilation was started with 1% enflurane in a mixture of nitrous oxide in oxygen, and the trachea intubated under excellent conditions. Anaesthesia was maintained with the same inhalational agents as above with an inspiratory minute volume of 6 litres/minute. Her electrocardiogram, oxygen saturation, and blood pressure were monitored throughout the anaesthetic with no abnormalities. Neuromuscular blockade was monitored by transcutaneous train-of-four (TOF) and tetanic stimulation of the ulnar nerve, using commercially available electrolyte pads connected to a Rutter nerve stimulator with visual assessment of the response.

Surgery was completed 30 minutes after induction, but response to TOF or tetanic stimulation was absent so intermittent positive pressure ventilation was continued with oxygen, nitrous oxide, and 0.75% enflurane. One hour and 15 minutes after induction of anaesthesia, one twitch was seen with TOF stimulation and post-tetanic potentiation observed. The inspired enflurane concentration was reduced to 0.5%. Fifteen minutes later, 1.5 hours after induction, four twitches were seen with a T4 to T1 ratio of approximately 30%. Neostigmine 2.5 mg and atropine 1.2 mg were given, and the enflurane and nitrous oxide discontinued. There was no fade on TOF stimulation within 3 minutes, tetanic stimulation was sustained for 5 seconds, and there was no post-tetanic potentiation. The trachea was extubated and the patient was immediately able to cough and perform a sustained head lift. She had no signs of residual neuromuscular blockade during the recovery period, and the postoperative course was uneventful.

Discussion

Myasthenia gravis is an autoimmune disease with a prevalence of approximately 1 in 20 000 of the population; females under 40 years of age are the most susceptible. The

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majority of patients exhibit an IgG antibody to the acetylcholine receptor on skeletal muscle, which acts via a complement-mediated lysis to cause a depletion of these receptors on the muscle end-plates.[1] The effect of the disease varies from minor ocular symptoms to generalised weakness and respiratory failure. Gradual deterioration usually occurs for the first few years, with frequent relapses and remissions. The disease may then enter a static phase with widely differing residual disabilities and need for treatment. Despite modern therapy, only 11% of generalised myasthenics go into true remission and become asymptomatic without treatment.[2]

It is interesting to speculate on the functional receptor population in symptomless myasthenic patients. Animal studies have shown a normal TOF response with 70% of the acetylcholine receptors blocked by curare,[3] and patients with a T4 to T1 ratio of 50% have adequate clinical muscular function,[4] so it is possible that symptomless myasthenic patients have a depleted receptor population. This would be reflected in their response to non-depolarising muscle relaxants since they have a reduced 'safety margin', normally provided by an excess of receptor sites. It is therefore likely that the concept of 'cure' does not apply to myasthenia gravis.

There are conflicting reports about the sensitivity to neuromuscular blocking drugs of myasthenics in remission. Brown and Charlton[5] studied sensitivity to curare in myasthenic patients using an isolated arm technique. They reported increased sensitivity regardless of the clinical state of the patient, including one patient whose myasthenia had been 'latent' for 15 years and who took no anticholinesterases. They also tested one patient during both a relapse and a remission and found very little difference in curare sensitivity. Lake[6] reported increased neuromuscular blockade due to curare during two anaesthetics in a patient taking only prednisone for myasthenia gravis. However, immediately before the first anaesthetic the patient had symptoms of weakness so cannot be considered to be in remission. Seven weeks later, when the patient had no neurological symptoms, a further anaesthetic using curare and enflurane again resulted in a greater than normal degree of neuromuscular blockade. Eisenkraft *et al.*[7] mention a case of increased sensitivity to atracurium in an asymptomatic patient on no therapy.

Conversely, Fillmore *et al.*[8] describe a patient with 'minimal symptoms' on prednisone alone who required normal doses of curare during enflurane anaesthesia. Azar,[9] in a review of neuromuscular disorders and muscle relaxants, concludes that myasthenic patients in remission respond normally to neuromuscular blocking agents, and that the sensitivity reported by Lake was due to the known potentiation of curare by enflurane.

Enflurane in normal subjects has a clinically significant effect on the neuromuscular junction. Enflurane, in the absence of neuromuscular blocking drugs, has no effect on peripheral twitch response at an alveolar concentration of less than 2%, but above this concentration the twitch response is depressed in a dose-dependent manner.[10] However, at lower concentrations enflurane potentiates non-depolarising blockers, again in a dose-dependent fashion. Gencarelli *et al.*[11] found that in the presence of a curare neuromuscular block 2.2% enflurane decreased twitch height to 8% of control. Keeping the curare levels constant they reduced the end-tidal levels of enflurane and demonstrated a remarkable improvement in twitch height, which recovered to 57% of control with 1.35% enflurane, and 91% with 0.5% enflurane. The potentiation of vecuronium by enflurane is thought to be less than that with curare or pancuronium.[12] It seems reasonable to suggest that in the normal patient vecuronium-induced neuromuscular blockade should not be greatly enhanced by the concurrent administration of enflurane at inspired concentrations of 1% or less.

The interaction between volatile agents and neuromuscular blockers is mainly due to a reduction in the chemosensitivity of the neuromuscular end-plate region by the volatile anaesthetics.[13] In myasthenia gravis this site is already depleted of acetylcholine receptors[1] so this interaction would be expected to be greater. Enflurane has been suggested as the sole anaesthetic agent in myasthenia gravis because of the easily reversible muscular relaxation obtained.[14]

The prolonged neuromuscular block seen in our patient was doubtless partly due to the potentiating effect of the enflurane. However, we felt it was right to continue to administer enflurane until a reasonable chance of reversal existed because of the possibility of awareness.

The potency of vecuronium was shown to be enhanced by hypercarbia.[15] No end-tidal CO_2 monitor was available at the time but since our patient was being ventilated at a minute volume of almost 90 ml/kg we consider that hypercarbia was not an influence in this case.

The accuracy of tactile assessment of TOF response has been questioned,[16,17]. but the method is used widely in clinical practice.

We suggest that symptomless myasthenic patients should not be regarded as 'cured' and, contrary to the opinion of Azar, they should be assumed to be sensitive to drugs that affect the neuromuscular junction, including non-depolarising neuromuscular blocking agents and volatile anaesthetics.

Acknowledgments

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Anaesthesia, 1989, Volume 44, pages 831–833

CASE REPORT

Spinal arteriovenous fistula

A possible cause of paraparesis after epidural anaesthesia

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Summary

A 62-year-old male suddenly developed a severe paraparesis after epidural anaesthesia. He recovered gradually over the next few months. He had an acute relapse one year later and a selective spinal angiography showed a dural T_8 arteriovenous fistula with large draining veins. Intravascular embolisation of the fistula produced immediate and sustained clinical improvement. The mechanism commonly held responsible for neurological disturbances in spinal dural arteriovenous fistulas is cord hypoxia secondary to venous hypertension. The 20-ml of local anaesthetic solution injected into a narrow spinal canal with osteophytosis may have caused further venous engorgement, cord hypoxia and acute neurological deficit.

Key words

Complications; paraparesis.
Anatomy; arteriovenous malformation.

We reported L_{3-4} spondylodiscitis as an unusual complication of epidural analgesia.[1] We have been very suspicious since then when the anaesthetic histories of patients referred to our Institute for lower limb sensorimotor deficits of unknown aetiology are considered. We have already described six cases of spinal arachnoiditis after epidural anaesthesia.[2] We now report a case of paraparesis after epidural anaesthesia in a patient with an arteriovenous fistula (AVF) of the spinal dura. This is, to our knowledge, the first described case in which spinal AVF is shown to be the cause of paraparesis after epidural block.

Case history

A 63-year-old male was admitted to our Institute in December 1987 because of a spastic hyperreflexic paraparesis. He had a bilateral inguinal herniorrhaphy and prostatectomy for fibroadenomatous hyperplasia under epidural anaesthesia 15 months earlier. He was ASA 2 because of mild arterial hypertension and asymptomatic mitral valve disease. Analgesia was obtained, after uneventful test dose, with a single dose of bupivacaine 0.5% 20 ml (100 mg) with adrenaline 1:200 000 injected at the L_{4-5} interspace. (The type of needle was not specified on the anaesthetic chart.) A loss-of-resistance technique with saline was used. There was no blood reflux from the needle.

Adequate analgesia was obtained for the entire surgical procedure (120 minutes). Slight hypotension, which did not require therapy, occurred during surgery. The patient claimed in the hours after the operation that 'the effect of anaesthesia didn't wear off completely' because of lower limb paresis and sensory impairment. A consultant neurologist confirmed the next day the presence of a severe spastic paraparesis with hypoaesthesia to all modalities from the groin downward. An indwelling urinary catheter was required for one month. The patient underwent physiotherapy. Neurological symptoms progressively improved and he was ambulant until 3 months before this admission when he experienced an acute relapse. He lost bladder control completely and could walk only for 4–5 metres with support. Moreover, he complained of a stinging pain in his feet, particularly when he attempted to walk. He had a sacral bed-sore when he was admitted to our Institute and, on neurological examination, spastic hyperreflexic paraparesis, global hypoaesthesia up to the groin, and urinary incontinence were present.

The patient also reported some irregular, minor difficulty in leg movements and paraesthesia when he exercised in the year before the herniorrhaphy but he paid no attention to these mild symptoms. Spinal X rays and magnetic resonance imaging showed a narrow lumbar canal with osteophytosis at L_{4-5} level. Examination of cerebrospinal fluid, obtained at myelography, showed an increase of protein concentration (0.72 g/litre; normal value 0.20–0.45 g/litre). Myelography (Fig. 1) raised the suspicion of a posterior dural arteriovenous fistula, which was demonstrated by

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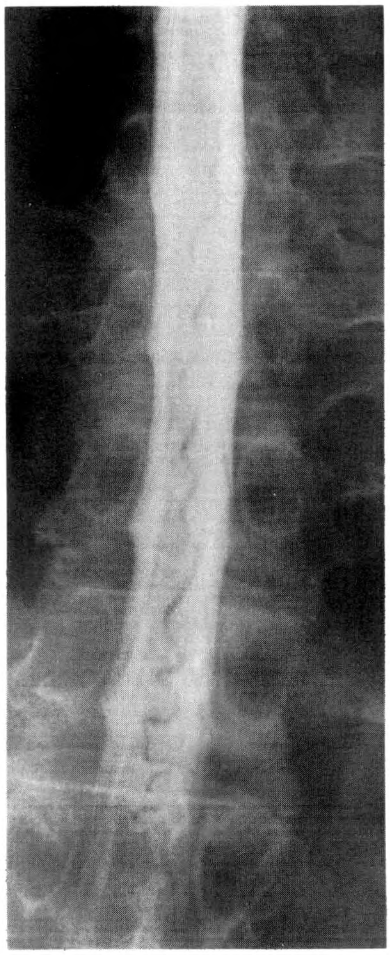

Fig. 1. Thoracolumbar myelogram. Anteroposterior view which demonstrates the typical appearance of the enlarged vessels in the retromedullary subarachnoid space.

selective medullary angiography; the fistula, located at the T_8 level, was accompanied by engorged, large veins (Fig. 2). The patient had a therapeutic intravascular embolisation of the fistula with calibrated particles of polyvinyl alcohol (Ivalon). An immediate improvement was obtained and the pain disappeared when the feeding vessel was occluded. Motor and sphincter functions showed a further mild recovery over the next few months.

Discussion

Paraplegia after epidural analgesia is a very rare complication with different causes.[3–5] The epidural anaesthesia in our patient appears to have precipitated an intermittent neurological disturbance, due to a spinal dural arteriovenous fistula, into a severe chronic paraparesis. Kendall and Logue, in 1977, first distinguished dural AVFs among the angiomatous malformations of the spinal cord.[6] They are relatively uncommon and their origin, whether acquired or congenital, is still disputed.[7,8] Their clinical manifestation is after the age of 40 years in 85% of cases.[7] The spinal cord

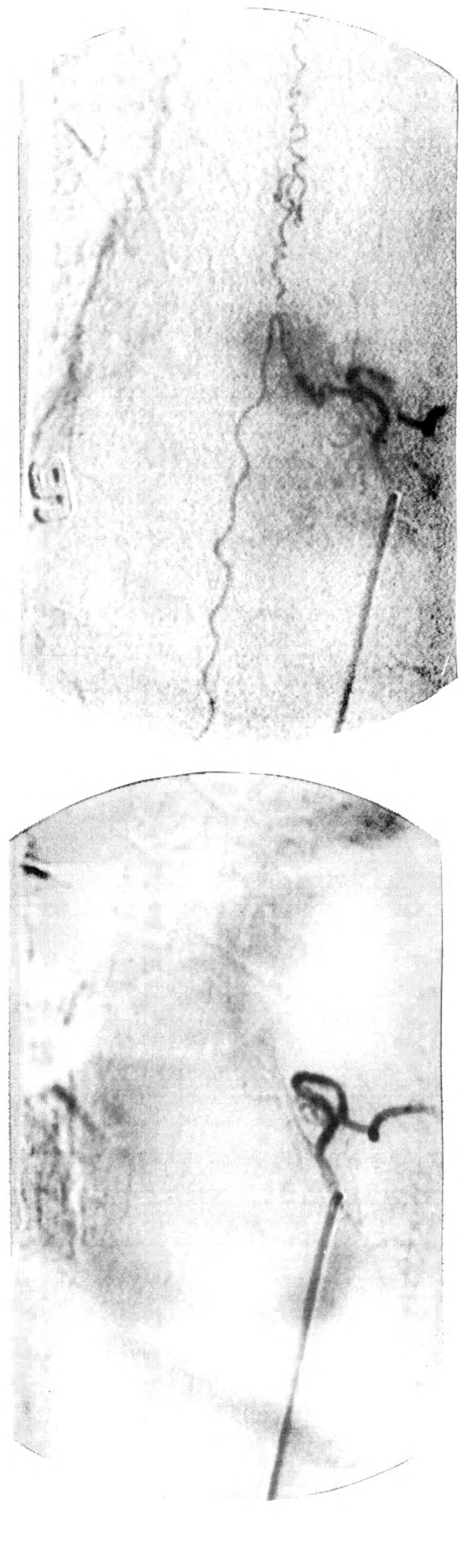

Fig. 2. *Above:* Anteroposterior spinal arteriogram which shows the enlarged veins with one feeding vessel which arises from the eighth left intercostal artery. *Below:* After embolisation: the posterior spinal artery is obstructed but, as desired, the intercostal artery is preserved.

symptoms are caused[6,7] by ischaemic hypoxia secondary to venous hypertension around the cord; hypertension results from the presence of the arteriovenous shunt and from impaired venous return, which in turn is probably caused by the absence of the veins normally draining into the epidural space.[7]

Our patient displayed, before and after surgery, intermittent weakness and pain, which are features of medullary claudication. This is one of the most frequent clinical presentations in the overall progressive course of spinal dural AVFs.[7] Neurological deficits were precipitated by the anaesthetic procedure, firstly because volume of 20 ml anaesthetic solution was injected into a narrow spinal canal. Secondly, osteophytosis restricted interlaminar foramina and hampered the diffusion of the solution towards paravertebral spaces.[9,10] Finally the veins which drain the AVF and medullary venous system are always grossly dilated in these cases[7] and behave like space-occupying structures.[9,11,12] All these events can cause ischaemic hypoxia of the spinal cord because of raised venous pressure. Intra-operative hypotension, even if slight, and the use of vasoconstrictors during the anaesthetic could also have acted as ischaemic agents.[11] This view is supported by the absence of clinical symptoms and signs of subarachnoid haemorrhage, epidural haematoma, arterial occlusion or intraspinal infection; these are, moreover, not usually reported as the presenting features of spinal dural AVFs.[7,8]

This case in our opinion, together with the other complications of epidural anaesthesia, suggest caution before prescription of a procedure which is generally considered to be safe. It also offers an interpretation of the neurological deterioration observed in patients with spinal AVF submitted to myelography.

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Anaesthesia, 1989, Volume 44, pages 834–836

CASE REPORT

Awake fibreoptic intubation for a rare cause of upper airway obstruction—an infected laryngocoele

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Summary

The anaesthetic management of a patient with an infected laryngocoele is presented. The relevance of this condition to the anaesthetist is discussed.

Key words

Intubation, tracheal; fibreoptic.
Complications; laryngopyocoele.

A laryngocoele is an abnormal dilatation of the ventricular saccule of the larynx. Baron Larey, Napoleon's surgeon, is credited with the first detailed clinical observation on laryngocoeles in 1829, and Virchow with the original description of their pathological features in 1867. Their origin is thought to be either a vestigial remnant that corresponds to the lateral air sacs found in lower animals, or an acquired laryngeal hernia (with or without congenital predisposition) that results from chronically elevated intralaryngeal pressure.[1]

Case history

A 42-year-old male machine operator was referred by his general practitioner to the ENT Department with a 6-day history of sore throat and neck pain, increasing dysphagia, an enlarging mass on the left side of his neck and a 2-day history of hoarseness. There were no other respiratory symptoms. Indirect laryngoscopy revealed, on initial examination, oropharyngeal inflammation and a swelling in the left hypopharyngeal region; both vocal cords appeared normal. Examination of the neck revealed a firm, tender, nonmobile swelling that measured 5×6 cm and lay in the anterior triangle of the neck. Laboratory investigation revealed a raised white cell count 13.1×10^9 cells/litre, and radiographs showed a fluid-filled sac that lay in the left side of the neck and caused slight deviation of the upper trachea and oesophagus, as shown in Figures 1 and 2.

The patient's condition deteriorated within 24 hours; as the size of the lesion increased the larynx markedly deviated to the right side of the neck, and dysphagia and increased hoarseness occurred. Indirect laryngoscopy then revealed a solid mass that obliterated the left pyriform fossa, and the left vocal cord could not be visualised. Urgent examination and biopsy under anaesthesia was requested.

Anaesthetic technique

The patient was given papaveretum 10 mg and scopolamine 0.2 mg intramuscularly one hour before operation as premedication. An intravenous infusion line for administration of antibiotics was sited in his left forearm.

The patient was pre-oxygenated while continuous electrocardiographic (Simonson and Weel Trioscope) and intermittent blood pressure recording (Datascope) took place. Anaesthesia was induced with 50% oxygen in nitrous oxide and halothane supplemented with incremental doses of methohexitone. Spontaneous breathing was maintained. There was evidence of airway obstruction, with increasing depth of anaesthesia, unrelieved by oro- or nasopharyngeal airways or changes of posture. Laryngoscopy was not attempted because the depth of anaesthesia was inadequate. The patient was therefore reawakened by stopping the anaesthetic and the substitution of 100% oxygen.

It was decided that the safest approach for airway control would be to use an awake fibreoptic intubation technique. Topical anaesthesia of 2 ml 10% cocaine was applied to the nasal mucosa using soaked cotton buds, and 2 ml 4% lignocaine hydrochloride via Forrester spray to the oropharynx, with the patient semi-sitting. The tip of the fibreoptic laryngoscope (Olympus LF-1) was introduced into the right nostril and slowly advanced. This revealed a

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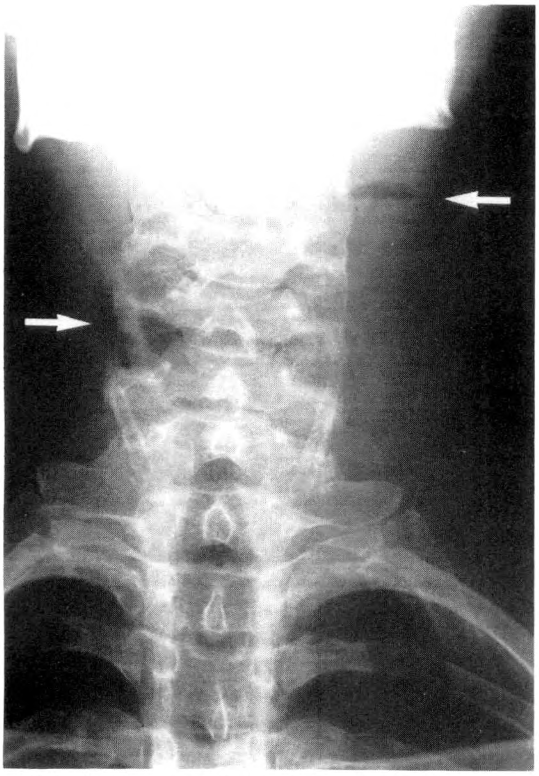

Fig. 1. Plain AP view of neck. An air–fluid interface is seen in the laryngeal sac on the left. There is deviation of the trachea and larynx to the right.

large polycystic lesion that arose from the left pyriform fossa with surrounding mucosal oedema and inflammation. The epiglottis was lying distorted and compressed to the right and only the right arytenoid and vocal cord could be seen. The tip of the laryngoscope was then advanced through an air bubble which lay across the cords, to allow entry into the trachea. There was no response from the patient. Further local anaesthesia (5 ml 2% lignocaine hydrochloride) was instilled via the side arm once the tip of the laryngoscope was in the trachea, which caused a slight cough. A red-rubber, cuffed nasotracheal tube, size 6, was introduced easily into the trachea using the fibreoptic laryngoscope as a guide. The position of the tube was checked visually by reference to the carina and by auscultation.

Anaesthesia was induced with methohexitone 50 mg, once the airway was secured, and muscle relaxation with atracurium 40 mg. Anaesthesia was maintained using intermittent positive pressure ventilation with oxygen 33%, nitrous oxide 67% and isoflurane. Surgery involved an external incision in the neck with dissection, drainage and laying open of a large laryngopyocoele.

Inspection at the end of the procedure showed only a residual swelling of the left supraglottis region and inflammation of an otherwise normal left vocal cord. Muscle relaxation was reversed with neostigmine 2.5 mg and glycopyrronium 0.5 mg. The patient was extubated fully awake when spontaneous respiration returned. There were no further airway problems.

Discussion

The incidence of laryngocoele in the UK is 1 per 2.5 million population per year. The sex distribution is 5:1 in favour of men and peak age incidence is 50–60 years. A laryngocoele can be external (30%) where the sac arises in the laryngeal ventricle and extends into the neck through the thyrohyoid membrane, internal (20%) where it arises and is confined to the laryngeal lumen, or combined (50%). Most are unilateral (85%) but some can occur bilaterally (15%).[2] The majority are asymptomatic but symptoms, if present, are related to the type of laryngocoele. They include hoarseness, due to impaired vocal cord movement, and stridor, due to encroachment of the lesion into the laryngeal lumen with airflow obstruction (both these symptoms are characteristic of an internal laryngocoele). There may be swelling of the neck anterior to the sternomastoid muscle (as seen with an external laryngocoele). There may also be symptoms of dysphagia, sore throat, snoring, pain or cough; 10% present with infected sacs—laryngopyocoeles. Symptoms tend to fluctuate and as with our patient may develop rapidly.[3]

There is debate about the association of laryngocoele and laryngeal cancer, and therefore direct laryngoscopy is

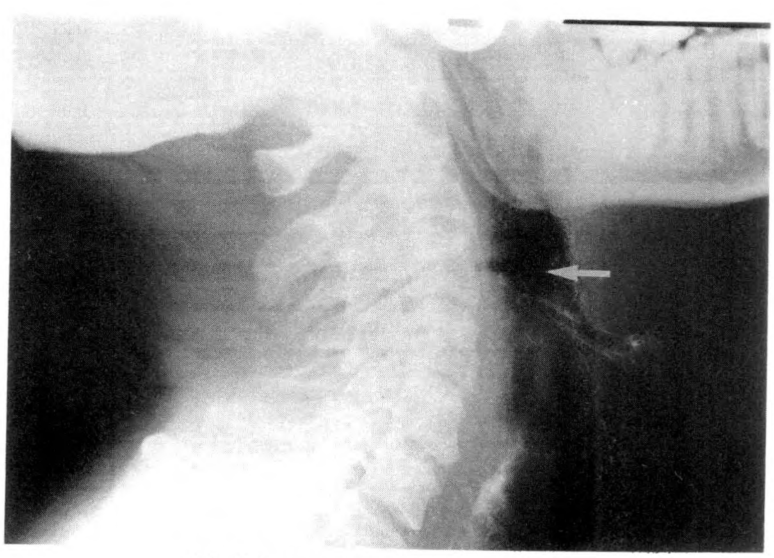

Fig. 2. Lateral view of the same patient.

mandatory to exclude coexistant pathology.[4] The diagnosis can usually be established from the history and radiographic examination, with plain anteroposterior and lateral X rays of the neck (with and without the Valsalva manoeuvres[5] which may aid inflation and identification of laryngeal air sacs). An air–fluid interface may be seen in laryngopyocoeles. We believe that in our patient laryngeal tomograms and computerised tomography may have helped outline the extent of the lesion and associated soft tissue involvement.

There are conditions and symptoms in anaesthesia which may make establishment of airway control and tracheal intubation difficult. These include musculoskeletal abnormality, stridor, hoarseness, dyspnoea, cyanosis and the presence of space-occupying lesions. However, unexpected difficulty may only become evident in some situations once the patient is anaesthetised. This may be due, when anaesthetised, to loss of tonic and phasic control of the airway. Previously these conditions either proved fatal or the airway was secured by tracheostomy with all its inherent risks.[6]

We suggest that an awake fibreoptic intubation technique with topical anaesthesia should be considered whenever any anatomical problem makes the success of securing the airway, using direct laryngoscopy, doubtful.[7] It is a safe, atraumatic and well tolerated procedure. Success with awake fibreoptic intubation depends on adequate preparation of the nasal, oropharyngeal and laryngeal mucosa with local anaesthesia and local vasoconstriction. Awake patients maintain muscle tone, can assist in clearing their own secretions and may aid visualisation of the larynx by phonation and tongue movement. The nasal route is preferred since the tongue is less likely to interfere with the procedure and the patient is unable to 'bite down' on the apparatus. Transtracheal injection of local anaesthetic is invasive, not essential for the provision of ideal conditions for intubation, is poorly tolerated and has an associated morbidity. Patient cooperation is essential and the use of sedation may compromise this.

Conclusions

All symptomatic cases of laryngocoele should be treated urgently, especially if there is associated infection, because life threatening symptoms may develop dramatically. An awake fibreoptic technique for airway control is recommended.

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Anaesthesia, 1989, Volume 44, pages 837–838

CASE REPORT

Postoperative hypotension associated with enalapril

R. M. RUSSELL AND R. M. JONES

Summary

We describe the case of a patient treated for hypertension with the angiotensin converting enzyme inhibitor enalapril, who developed hypotension after recovery from anaesthesia.

Key words

Pharmacology; angiotensin converting enzyme inhibitor, enalapril.
Complications; hypotension.

Angiotensin converting enzyme (ACE) inhibitors were introduced into clinical practice in 1981[1] and have been used subsequently with increasing frequency, for the treatment of hypertension[2] and congestive cardiac failure.[3] We report the case of a hypertensive patient treated with enalapril who became profoundly hypotensive after emergence from anaesthesia.

Case history

A 52-year-old female who weighed 63 kg was admitted for elective surgical management of left carpal tunnel syndrome. She was diagnosed as hypertensive one year before at a routine blood pressure check, and started on treatment with enalapril 10 mg daily. She had no symptoms related to her cardiovascular system. She was anaesthetised uneventfully on three previous occasions, most recently 4 years ago. She appeared fit on examination; her heart rate was 60 beats/minute and her blood pressure 120/70 mmHg. Her heart sounds were normal and her chest was clear on auscultation. Abdominal examination was unremarkable and a full blood count and serum electrolyte concentrations were within normal limits. She took her usual dose of enalapril 2 hours before she was scheduled for surgery. Premedication was with intramuscular papaveretum 15 mg and hyoscine 0.3 mg one hour before surgery. Blood pressure before induction of anaesthesia was 100/60 mmHg. A vein on the right hand was cannulated. Anaesthesia was induced with thiopentone 300 mg and maintained with 66% nitrous oxide in oxygen and enflurane 1–3%; the patient breathed spontaneously through a Mapleson D system. A tourniquet was applied to the left arm and inflated to 250 mmHg for 27 minutes during surgery. Intraoperative monitoring comprised continuous electrocardiography and noninvasive blood pressure recordings every 3 minutes. Blood pressure fluctuated between 85 and 95 mmHg systolic and 45 and 55 mmHg diastolic.

The patient was moved to the recovery area after surgery and breathed oxygen through a Hudson mask with a flow rate of 5 litres/minute. She was awake on arrival in the recovery area, was well perfused, with a heart rate of 75 beats/minute and a blood pressure of 120/80 mmHg. She remained awake and asymptomatic, but a further blood pressure measurement 15 minutes later was 50/30 mmHg; heart rate was 70 beats/minute. There was no blood loss or other factor which could account for hypotension. One litre of compound sodium lactate solution was infused rapidly, followed by 500 ml of polygeline; incremental doses of intravenous ephedrine were administered to a total of 15 mg. The blood pressure increased to 80/50 mmHg during the next 10 minutes and 30 minutes later was 95/55 mmHg. The patient was discharged back to the ward 2 hours after arrival in the recovery area; the blood pressure at this time was 100/50 mmHg and the heart rate 55 beats/minute. The blood pressure did not decrease below 90 mmHg systolic during the next 24 hours. The patient felt no ill effects, and was discharged home the next day.

Discussion

The reduction of blood pressure after administration of enalapril is due primarily to a decrease in peripheral vascular resistance caused by inhibition of the enzyme that converts inactive angiotensin I to the active vasoconstrictor angiotensin II.[4] This same enzyme also inactivates the vasodilator peptide bradykinin.[5]

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Enalapril is a prodrug absorbed rapidly after oral administration and hydrolysed to the active compound enalaprilat (enalaprilic acid), a highly specific, long-acting, nonsulphydryl ACE inhibitor. Enalaprilat binds to ACE to cause maximal inhibition for 10 hours; peak plasma concentrations occur approximately 3 hours after ingestion. Enalapril and enalaprilat are both excreted via the kidney. First doses of ACE inhibitors can cause a dramatic reduction in blood pressure over 3–4 hours, especially in patients who are taking concomitant diuretic therapy, and those with renovascular hypertension.

In this patient, blood pressure was maintained during surgery, but decreased when she regained consciousness in the recovery period. First-dose hypotension cannot be responsible because the patient was well controlled on the same therapy for more than one year.

One possible explanation for the sudden appearance of postoperative hypotension relates to inhibition by enalapril of the breakdown of bradykinin; there may have been accumulation of this and other vasodilator metabolites during surgery, possibly enhanced by the action of vasodilating anaesthetic drugs, and blood pressure may have been maintained during surgery by increased endogenous catecholamine concentrations. At the completion of surgery, catecholamine concentrations may have decreased to a level at which hypotension resulted. The reduction in blood pressure occurred when plasma concentrations of enalaprilat were likely to have been at their peak. The administration of intravenous fluids and a vasoconstrictor reversed the hypotension rapidly and effectively. Yates and Hunter[6] reported that the pressor response to sympathetic stimulation, such as may occur during laryngoscopy and tracheal intubation, is less in patients pretreated with enalapril and suggested that enalapril pretreatment may reduce postoperative hypertension. These observations are not necessarily at odds with our own report. Yates and Hunter studied normotensive patients who underwent painful abdominal surgery, whereas our patient was a known hypertensive who was subjected to a much less painful peripheral procedure. We would not disagree that, in some circumstances, deliberate treatment with enalapril may promote intra- and peri-operative haemodynamic stability. However, we suggest that blood pressure should be monitored carefully in hypertensive patients who are on long-term treatment with enalapril, especially if relatively minor surgery is performed, and that monitoring should be continued into the postoperative period when the stimulation of surgery has ceased.

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CASE REPORT

Minitracheotomy—a life-threatening complication

A. K. DABORN AND M. N. E. HARRIS

Summary

A 55-year-old patient developed profuse haemorrhage immediately after insertion of a minitracheotomy tube. Measured blood loss was 1.1 litres, and the bleeding required to be controlled surgically. The cause was a subglottic granuloma which had developed after prolonged tracheal intubation and which was incised during insertion of the minitracheotomy.

Key words

Equipment; minitracheotomy.
Complications; haemorrhage.

Percutaneous minitracheotomy packs have been available since 1983. They are used to provide emergency access to the airway, or to provide a route for removal of secretions and assistance with physiotherapy in patients who might otherwise require formal tracheostomy. This case report describes life-threatening haemorrhage into the airway after insertion of a minitracheotomy tube.

Case history

A 55-year-old woman presented for coronary artery bypass surgery. She had suffered from severe hypertension for more than 10 years (up to 240/100 mmHg despite treatment), and incapacitating angina. This was secondary to her familial hypertriglyceridaemia and maturity onset diabetes. The patient had suffered from angina for more than 3 years. The anaesthetic technique which was used during her triple coronary artery graft procedure has been described elsewhere in detail.[1] She was not rousable after the procedure and spontaneous respiration was inadequate. Doxapram and flumazenil were administered, but resulted in little improvement and so ventilation was continued postoperatively. The lungs were ventilated with volume-controlled ventilation for 8 days; subsequently, continuous positive airway pressure was used via the tracheal tube. This continued pressure support was required because of intercurrent chest infections that caused atelectasis and poor gas exchange. Her neurological condition improved but gross deficits remained. Computerised axial tomography showed bilateral occipital infarcts with marked cerebral oedema.

Her trachea was extubated 16 days after surgery. Her ventilatory drive was good, but she had mild stridor which worsened over the next 12 hours. The oxygen saturation remained at 95–99% and the $Pa\text{CO}_2$ at the end of this period was 6.5 kPa. Her respiratory rate had increased from 20 to 35 breaths/minute. A nasopharyngeal airway was inserted after 20 hours and shortly afterwards her condition deteriorated rapidly. Sternal and intercostal recession were marked and her $Pa\text{CO}_2$ increased to 8.5 kPa. Her trachea was re-intubated without paralysis and her clinical condition improved rapidly. The intubation was moderately difficult but this was explained by the absence of relaxant. Direct laryngoscopy had shown that the tip of the nasopharyngeal airway was close to, and possibly stimulating, the larynx with each inspiratory effort, but the larynx appeared entirely normal. Mucous secretions were spilling from the larynx. The decision was made to insert a minitracheotomy because this would allow adequate removal of secretions as well as increasing the available effective diameter of the airway to relieve the stridor, which was assumed to be the result of either laryngospasm or pseudobulbar palsy.

The equipment used was the Portex Mini-Trach II kit. The patient was supine with a small pillow under her shoulder. This gave good access to her cricothyroid membrane, which was easily identified. The skin was cleaned and infiltrated with lignocaine and adrenaline before insertion of the guarded blade. The skin was tethered on either side of the cricoid cartilage with finger and thumb to prevent separation of the layers after removal of the blade. The introducer clearly entered the cricothyroid membrane, but it proved very difficult to slide the Mini-Trach tube over it. Three attempts were made to achieve

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this. Just before the third attempt, coughing and bleeding from the patient's mouth became apparent. The tube was inserted, but only blood came from its lumen. Ventilation became inadequate and the oxygen saturation dropped rapidly to 75%.

Emergency re-intubation was attempted. The pharynx and mouth were filled with fresh blood and clots. Initially, suction was inadequate to allow visualisation of the larynx. Eventually the profuse haemorrhage was seen to come from below the vocal cords. The patient's condition dramatically improved after tracheal intubation, but the oxygen saturation had decreased as low as 37% and hypoxic bradycardia had occurred just before re-intubation. The bleeding continued over the next 2 hours; the total loss was 1100 ml and blood transfusion was required.

Direct laryngoscopy and bronchoscopy, before definitive tracheostomy, revealed a large subglottic granulomatous polyp which occluded more than half of the tracheal diameter and which extended for 3–4 cm down the trachea. Histological examination confirmed it to be a granulomatous polyp. The haemorrhage had occurred from damage to this lesion by the tip of the Mini-Trach tube or introducer and the bleeding was controlled surgically. A tracheostomy tube was inserted and ventilation continued for a further 8 hours.

Discussion

Direct access to the trachea and airways via the cricothyroid membrane is now well established. It was described initially in 1921[2] but its use was condemned and this detracted from further evaluation until a report in 1976[3] showed fewer complications from this route than from formal tracheostomy. Chronic subglottic stenosis was the major complication described, but this followed insertion of a standard tracheostomy tube. Further evaluation of subglottic stenosis in 1982 resulted in the recommendation that the procedure should be reserved for adults whose trachea had been intubated for less than 10 days and who had no laryngeal pathology.[4] Laryngoscopic examination of the chronically intubated patient was recommended before cricothyroidotomy because obstruction of the subglottic space after all forms of airway intubation was often caused by granulation tissue.

The introduction of the Portex Mini-Trach in 1983 followed the description of its use for suction access to the airway.[5] Its success for this indication in the absence of tracheal intubation or formal tracheostomy has been well documented.[5–7] Reports of serious complications have been few: inhalation of the original tube after breakdown of skin sutures,[8] or inadequate care during sputum aspiration,[9] were potentially lethal problems solved by the introduction in 1985 of the Portex Mini-Trach II. External bleeding and emphysema are possible, but considered to be minor complications;[10] the authors of this report were confident enough to suggest that minitracheotomy insertion could be performed even during anticoagulant therapy. Difficulty with insertion or misplacement of a minitracheotomy after its use for high frequency jet ventilation has also been documented.[11] External bleeding is common, and minimised by the use of a vasoconstrictor with the local anaesthetic. Severe external haemorrhage can be envisaged only if an incorrect site of incision is used or if aberrant thyroid tissue or blood vessels exist. There is only one previous report of severe bleeding into the airway after insertion of a Mini-Trach II[12] but the cause for this bleeding was not identified.

This patient had been anticoagulated with warfarin for several days before the insertion of the minitracheotomy tube. Anticoagulation is not a recognised contraindication to this procedure, and it is unlikely that the coagulation status is of great importance with this sort of rapid haemorrhage. However, it must be regarded now as another factor to consider when the decision is made to insert a minitracheotomy tube.

Decreased infection risk in the sternotomy wound of a cardiac surgical patient by avoidance of formal tracheostomy is considered desirable.[7] This heavily influenced our decision to use a minitracheotomy in this patient. Despite prior laryngoscopy with a standard Macintosh blade, we were unable to see the subglottic granuloma. We therefore mistakenly assumed that the nasopharyngeal airway was involved in causing the stridor. Our experience with this patient has demonstrated that minitracheotomy is not an uncomplicated procedure. The outcome would certainly have been fatal if facilities for rapid and difficult intubation and resuscitation had not been available. This case underlines the importance of awareness that major haemorrhage may occur after minitracheotomy if a subglottic granuloma is present. The procedure is indicated often in patients who have already been submitted to a period of conventional ventilation, and so the possibility of a subglottic granuloma is a very real one. Consequently, we advise insertion of a minitracheotomy tube only in a theatre or intensive care area in order to assure immediate access to these facilities. We support the view that minitracheotomy can be used safely even after a prolonged period of intubation[13] only if these recommendations are followed.

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CASE REPORT

Emergency resection of phaeochromocytoma presenting with hyperamylasaemia and pulmonary oedema after abdominal trauma

D. J. GREAVES AND P. M. BARROW

Summary

Phaeochromocytoma may present as an acute emergency with a perplexing variety of symptoms. We report a case in which a tumour of the organ of Zuckerkandl was removed after its unexpected discovery during laparotomy for abdominal trauma. A patient is described in whom a history of abdominal trauma coupled with collapse, pulmonary oedema, raised serum amylase and a positive peritoneal tap for blood, led to laparotomy at which an extramedullary phaeochromocytoma was found unexpectedly. The tumour was successfully resected, but immediate hypotension was life threatening. The inadvisability of resecting a phaeochromocytoma discovered at operation is discussed.

Key words

Surgery; acute, phaeochromocytoma.
Complications; pulmonary oedema.

Case history

An altercation outside a public house on New Year's Eve culminated in the collapse of a 22-year-old policeman who was bear-hugged by a reveller. He was pale and sweaty on admission with poor peripheral perfusion; he complained of discomfort in the back and was tender in the epigastrium. His pulse rate was 130 beats/minute and blood pressure 110/60 mmHg.

The serum amylase was 3600 IU/litre on laboratory investigation. A diagnosis of traumatic pancreatitis was made and the patient admitted for observation.

Five hours after the assault, the patient became dyspnoeic, cyanosed and coughed up frothy bloodstained sputum. Auscultation of the chest revealed coarse crepitations throughout both lungs. Blood pressure was 100/70 mmHg. Chest X rays showed a butterfly-wing distribution of shadowing, characteristic of pulmonary oedema. Blood gas determination on 60% inspired oxygen was: $Pa\text{O}_2$ 5.6 kPa, pH 7.35, $Pa\text{CO}_2$ 4.35 kPa, base excess −6.4 mmol/litre, O_2 saturation 75%. The central venous pressure (CVP) was 5 mmHg at the mid axillary line. The straight abdominal X ray showed an appearance that was reported as possibly due to retroperitoneal gas. Ultrasound scan of the pancreas showed no abnormality, but a guided aspiration of an echo in the suprapubic area yielded frank blood.

An intra-abdominal catastrophe was suspected and urgent laparotomy was undertaken because a ruptured duodenum seemed a strong possibility. The cause of the pulmonary oedema was unclear but adult respiratory distress syndrome, secondary to abdominal trauma, was considered likely.

Operative and anaesthetic management

A radial arterial line was inserted before operation. Induction of anaesthesia was carried out with ketamine 110 mg followed by suxamethonium 100 mg. The blood pressure immediately increased to 270/150 mmHg, and on intubation large quantities of oedema fluid came out of the tracheal tube. Vecuronium was given to facilitate ventilation and 50% nitrous oxide in oxygen was supplemented with diamorphine.

The pancreas and duodenum were normal at laparotomy. A very vascular tumour 10 cm in diameter was lying over the bifurcation of the aorta. It seemed probable that the tumour was an extra adrenal phaeochromocytoma and it was agreed with the surgeon that in view of its accessibility, he should resect it.

The patient's condition improved and the blood pressure was now 130/100 mmHg. A litre of plasma protein fraction (PPF) was rapidly infused and catecholamine infusions

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were prepared. The systolic arterial pressure decreased to 20 mmHg when the blood supply to the tumour was clamped. The CVP decreased to 1 mmHg and raised ST segments were seen on the ECG. Fast infusion of PPF 1 litre and 0.9% saline 2 litres was accompanied by catecholamine support. Initially adrenaline and isoprenaline were used. Despite increasing inotrope support (adrenaline 0.6 μg/kg/minute and isoprenaline 0.2 μg/kg/minute) the systolic arterial pressure remained low (30 mmHg) and a bradycardia (20 beats/minute) developed. A bolus of adrenaline 0.5 mg was given, which raised the systolic arterial pressure to 80 mmHg and the heart rate to 130 beats/minute. Adrenaline, at 1.5 μg/kg/minute, maintained the systolic arterial pressure at 80 mmHg. Surgery was completed and the abdomen closed. Spontaneous ventilation was re-established and the subsequent Pa_{O_2} on 40% oxygen by mask was 12 kPa. A postoperative chest X ray within 2 hours showed resolution of the pulmonary oedema. It proved possible to begin reduction of the catecholamine support immediately and it was stopped 12 hours after the operation.

The patient progressed well. Histological examination of the specimen showed appearances typical of an extramedullary phaeochromocytoma of the organ of Zuckerkandl. A pre-operative urine sample from our patient indicated a tumour with mixed secretions. Noradrenaline 2118 ng/ml (normal range 60–650 ng/ml) adrenaline 456 ng/ml (normal range 10–200) and dopamine 506 ng/ml (normal range 280–2300).

Discussion

Phaeochromocytoma can present as an acute emergency in many ways[1] and minor trauma has been described as a cause of catecholamine crisis.[2] Acute abdominal symptoms may predominate.[3] Cardiac complications including pulmonary oedema[4-6] may occur. Raised serum amylase has been observed,[7] as has the combination of pulmonary oedema and hyperamylasaemia as seen in our patient.[8]

Much has been written on the correct choice of anaesthetic agents for elective resection of phaeochromocytoma.[9] The technique was chosen for this patient in the belief that the clinical state reflected haemorrhagic shock, and agents were used that are unsuitable for a patient with a phaeochromocytoma. Suxamethonium may stimulate the sympathetic ganglion and fasciculations may squeeze the tumour.[10] Ketamine may produce hypertensive episodes and is a bad choice of induction agent.[11] Induction of anaesthesia precipitated severe hypertension and worsening of pulmonary oedema.

Rebound hypotension on removal of the tumour is to be expected in a patient who has not been suitably pretreated, both from reduction in catecholamine drive on the heart and from peripheral vasodilatation. Inotropes and rapid transfusion to restore the circulating volume are needed. Very aggressive pharmacological support was required to restore a satisfactory blood pressure in this patient, and time was wasted progressively increasing the doses of adrenaline and isoprenaline to no effect. Only a large bolus followed by infusion of adrenaline at a very high rate was found to be adequate.

Elective resection of phaeochromocytoma with pre-operative pharmacological preparation carries a low mortality, whereas anaesthesia in undiagnosed unprepared patients is very hazardous.[12-14] Conservative treatment and subsequent elective operation are the treatment of choice when the diagnosis of phaeochromocytoma has been made pre-operatively. Emergency resection of recognised phaeochromocytoma is never indicated.[9] It is tempting to proceed as we did with resection when confronted with an accessible tumour in a patient at laparotomy, but a better course of action would have been carefully to close the patient and begin treatment.[12,15] Control of hypertension with sodium nitroprusside can be followed by the introduction of α adrenergic blockade. A planned resection could then be made at a later date.

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HISTORICAL

Two early ether inhalers

A. H. B. MASSON

Summary

The existence of two of the earliest ether inhalers, a Squire's inhaler and a Tracy's inhaler, in the Museum of the Royal College of Surgeons of Edinburgh is recorded.

Key words

History; diethyl ether.

The news of the discovery of ether anaesthesia reached Britain on 17 December 1846 in a letter from Professor Jacob Bigelow in Boston to his friend, Dr Francis Boott in London. Boott and a London dentist, James Robinson, spent that day and the next 'rigging up' an inhaler which consisted of two glass containers, one above the other, originally the upper and lower part of a Nooth's apparatus.

Nooth's apparatus was designed for making soda water. The lower chamber, when used for that purpose, was filled with marble chips and sulphuric acid, while the upper contained the water to be carbonated. Pieces of sponge 'previously moistened with one ounce of ether' were put into both chambers, when it was adapted for ether inhalation. A tube from the lower chamber to the mouthpiece had a stopcock, a 'perpendicular valve' which opened on inhalation and closed on exhalation, and a 'horizontal valve' through which exhaled gases escaped. There was also a 'nasal spring' to compress the nostrils.

It was, they admitted, 'a very imperfect apparatus, hastily got up' but they used it, with success, on a Miss Lonsdale on Saturday, 19 December. However, they were not so successful the following day when they tried, and failed, to produce insensibility in three or four cases. Boott attributed the failure to a defect in the valve of the mouthpiece. They consequently improved the design. Their apparatus, subsequently constructed 'on similar principles, but of more elegant form' by 'Mr Hooper, Operative Chemist, of Pall Mall East', is known as the Robinson or the Hooper inhaler.[1]

The Squire inhaler

The surgeon, Robert Liston immediately went to see Boott when he heard of his success and took with him a young medical student, William Squire. Their first experiments were not successful. 'The glass vessel was too small', so they tried a 'sponge alone covered with a folded cloth', with more success. Liston then took young Squire to Oxford Street to the premises of Squire's uncle, Peter, an instrument maker who was a friend of Liston's. Here, ether was given by means of a sponge to 'one of the assistants'. However, because of the 'strong smell of vapour in the room, the necessity for a glass or other vessel to contain the ether was obvious'. Squire senior 'became much interested and with his energetic assistance a suitable inhaler was improvised'—the Squire's inhaler.[2]

Liston arranged to perform an amputation on the Monday (21 December), with William Squire's assistance. Squire took the inhaler—a 'glass vase' and 'a supply of ether prepared by my uncle' to the hospital. He had the apparatus brought close to the left side of the patient's head and administered ether to Frederick Churchill.

It is not surprising, since William Squire went straight from Boott's house to his uncle's premises, that the 'ingenious apparatus extemporaneously contrived by Mr Squire of Oxford Street' was very similar to that used by Boott and Robinson. It too was essentially a Nooth's apparatus, with a valve above the upper chamber to admit air. Dr Forbes, a witness to Liston's historic operation, wrote: an inhaler 'on the same general plan as Mr Robinson's but with what we consider an improvement in the mouthpiece, and in the regulating valves, has been introduced by Mr Squire'.[3]

Liston promptly reported his success to, amongst others, his old friend and former assistant, James Miller, who was then Professor of Surgery at Edinburgh University. In his jubilant letter to Miller, he wrote: 'Shall I desire Squire, a most capital and ingenious chemist, to send you a tool for

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Fig. 1. Squire's inhaler.

the purpose? It is only the bottom of Nooth's apparatus with a sort of funnel above, with bits of sponge, and, at the other hole, a flexible tube'.[4] Miller accepted the offer and the apparatus Liston sent was used for Miller's first operation under anaesthesia on 17 January 1847.[5]

There were certainly some practical difficulties associated with the Robinson and the Squire inhalers. The Nooth's apparatus, upon which they were based, is cumbersome, fragile and rather unstable since it consists of several glass containers balanced, somewhat precariously, one on top of the other. They both had pipe mouthpieces and valves which did not work well if the patient was not sitting upright.

Many inhalers were devised, but none of the early versions gained general acceptance. Squire's was 'much used' and Robinson's, probably the most popular, 'has at least the merit of success in its favour',[3] but the use of most of them was discontinued within a few months.

The original inhalers were not popular in Edinburgh. James Young Simpson seldom mentioned inhalers other than the one he devised. On the occasion of his first use of anaesthesia, he stated merely that he 'made the patient inhale the ether vapour'. Simpson reported to the Medico-Chirurgical Society as early as 3 March 1847, that he was using an 'oval flask' of his own design with no valves.[6] He wrote that 'when it is not necessary to keep up the etherization above five or ten minutes, by far the best and most perfect inhaler was formed by a large sponge'. However, 'when a prolonged effect is required, as in midwifery cases, an instrument is necessary—were it for no other reason than the saving of ether, and the prevention of its diffusion through the apartment'.[7]

Miller, too, very soon abandoned the 'very beautiful yet simple apparatus made by Squire' in favour of an ether-soaked sponge. He clearly had some problems with early inhalers. He complained, as have many surgeons over the years since, of 'the loss of time which the imperfect exhibition of ether must ever occasion in private practice'. He also observed that 'dentists everywhere were beginning to rue greatly their loss of time by ether; but now that its exhibition had been simplified and improved (and in London taken up as a distinct profession), the objection on that score had been in great part, if not altogether removed'.[8]

The irascible and bigoted James Syme gave up the use of ether for a time 'until more certainty could be reposed on its administration'. That came about when he was persuaded to use the 'very simple and efficient apparatus which was said to have been extensively used in obstetric practice by the Professor of Midwifery' (i.e. Simpson). 'The wide openings and absence of valves render respiration by this apparatus perfectly free'.[9]

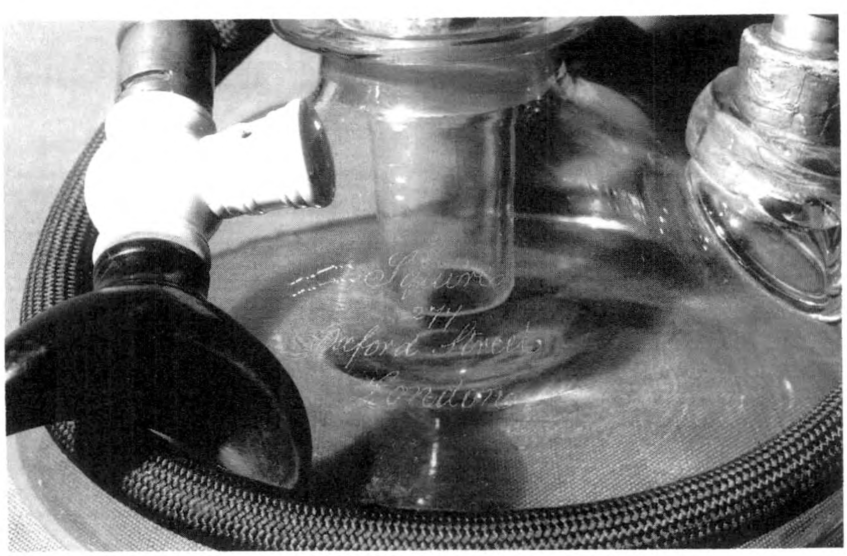

Fig. 2. Manufacturer's name on Squire's inhaler.

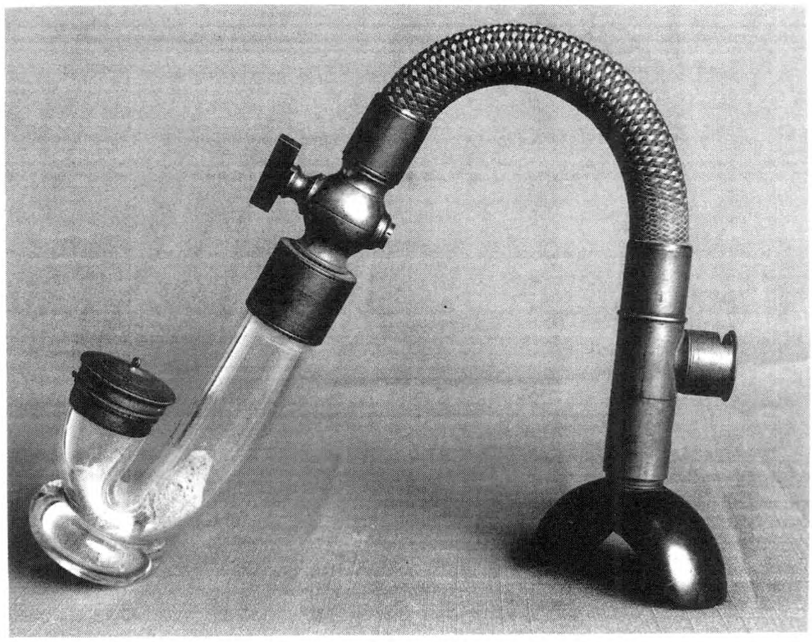

Fig. 3. Tracy's inhaler.

The popularity of the more complicated ether inhalers, therefore, was never great in Edinburgh. The three dominant figures in the city, Simpson, Miller and Syme, had all abandoned their use within a month or so. It is remarkable, therefore, that there is a Squire's inhaler in the Museum of the Royal College of Surgeons of Edinburgh (Fig. 1). The maker's name and address, 'Squire, 277 Oxford Street London' are clearly engraved on the upper chamber (Fig. 2) and the whole instrument is in excellent condition.

The Tracy Inhaler

There is also a Tracy inhaler (Fig. 3) in the same museum and in equally good condition. It is a hookah-shaped glass with a 41 cm elastic tube that leads from the glass to the mouthpiece. Its originator, Dr S. J. Tracy, of St Bartholomew's Hospital in London, wrote[10] in January 1847, 'I could not but observe the inconvenient size and costliness of the various apparatus, combined with the expense of preparing them for use'. He devised an apparatus which, he said, combined simplicity with utility and had it made for him by the Instrument Maker to the hospital, a Mr Ferguson.

The Tracy inhaler, too, was soon superseded. Tracy himself gave it up in August 1847, in favour of a saturated sponge 'having heard that Mr Miller of Edinburgh had used it (ether) successfully in that form'. The Edinburgh Tracy, however, was not made by Ferguson. The maker's name, 'Kemp & Co' is engraved on the brass stopcock (Fig. 4). Kemp and Company was an Edinburgh firm, established in 1835, which made laboratory supplies and scientific instruments.

The question of how these inhalers came to be in the possession of the Royal College is intriguing but, unfortunately, cannot be answered. There is no record of how, or when, this occurred. However, we know for certain that Liston sent a Squire's inhaler to Miller and there must be more than an even chance that that is the inhaler in question.

Other people, Simpson for instance, might also have obtained one. It is known that Simpson went to London in 1846 'during the Christmas holidays' and that he saw Liston. They obviously discussed the great event and Simpson 'went back from this London visit to Edinburgh bent on testing its applicability to midwifery'. However, it seems unlikely that he brought back an inhaler with him. It

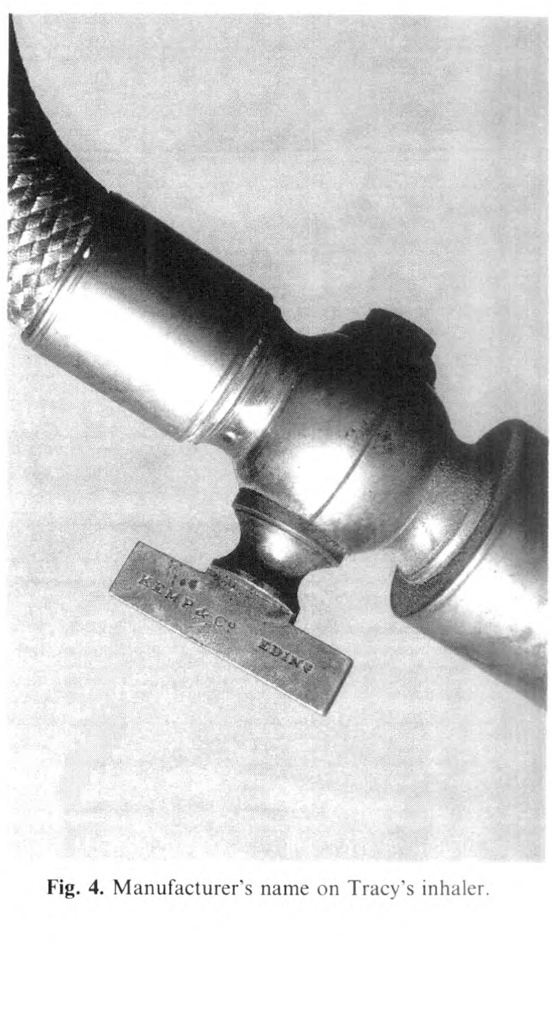

Fig. 4. Manufacturer's name on Tracy's inhaler.

would have been quite out of character for him to have had an inhaler in his possession and not to have used it for 3 weeks. He did not even see ether inhaled until January 1847[11] and he did not use it until 19 January of that year, after both Duncan and Miller had operated under ether. His real conversion to the benefits of anaesthesia came after he had used ether himself in an obstetric case, when he wrote 'I can think of naught else'.[12]

Tracy inhalers are rare but the Squire inhaler may be unique. The Science Museum in London, which houses the Wellcome Museum of the History of Medicine, has a replica but does not have an original. The curators stated: 'We are not aware of there being any others in major British collections, but we have not sought to investigate its rarity'.[13] Whatever its origin, therefore, the one in the Royal College of Surgeons of Edinburgh is of considerable interest. Any information about other specimens would be welcomed.

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Anaesthesia, 1989, Volume 44, pages 847–850

HISTORICAL

Paul Sudeck—his contribution to anaesthesia

D. D. C. HOWAT

Summary

Paul Sudeck is not generally recognised as a pioneer in anaesthesia, although he is well known for the atrophy of bone named after him. However, he not only championed the use of ether as a safe anaesthetic agent, described a method of ether analgesia for outpatient surgery and devised an inhaler for its administration, but also reintroduced nitrous oxide into Germany and invented possibly the first circle carbon dioxide absorption system with an optional attachment for continuous positive pressure respiration.

Key words

History; Paul Hermann Martin Sudeck.

Sudeck is known best to British doctors for his description of the atrophy of bone which may occur after fractures, particularly at the wrist, and for his 'critical point' on the pelvic colon, the site of the anastomosis between the sigmoid and superior rectal arteries. His great interest in general anaesthesia, however, is not so well recognised.

Paul Hermann Martin Sudeck was born in Pinneberg in Schleswig-Holstein, north of Hamburg, on 28 December 1866, the year of the Austro-Prussian War which established the hegemony of Prussia and the beginning of the unification of Germany. He came from a family of lawyers and his father was a Councillor of the Supreme Court of Hamburg. Paul became a citizen of that city in 1881 at the age of 15 years and, at 24 years, a Bürger or Freeman. He attended, after school in Altona in Hamburg, the Universities of Tübingen, Kiel and finally Würzburg, where in 1889 he was awarded the degree of Doctor of Medicine after presenting a thesis on poisoning by potassium chlorate. He looks somewhat severe in the photograph (Fig. 1), but he seems to have been a modest man. He points out in several places that his findings on ether and nitrous oxide were not new and had been described by many writers from 1847 onwards. He gives full recognition to his assistant, Helmut Schmidt, in his papers on nitrous oxide and on his anaesthetic machine, and handed over the description of the apparatus to him, because of his expertise in technical and practical investigation.

He completed 3 years as assistant in the Institute of Pathology in Würzburg, and returned to Hamburg. Here, after assistantships in medicine and surgery, he climbed the surgical ladder to become the equivalent of Senior Lecturer or Reader, with the title of Professor in 1919, in the new University of Hamburg, of which Hermann Kümmel was the first Professor of Surgery. Sudeck succeeded him as full Professor in 1923, at the age of 57 years. He had a wide interest in medicine and surgery, which included neurology, urology and especially orthopaedics.[1] He became interested in ether anaesthesia, particularly for short operations whilst working as Kümmel's assistant in the Eppendorf Hospital in Hamburg. Over 20 years later, as Professor of Surgery, he was responsible for reawakening interest in nitrous oxide in Germany, where it had not been in use for many years and, with his assistant Schmidt, designed an anaesthetic machine with a circle absorber circuit and an attachment for positive pressure anaesthesia. Sudeck is described in the University records as well-known for his careful surgery, his considerable appreciation of the arts and his keen interest in anaesthesia.[2] He retired in 1935 and died in Saalfeld in Thuringia on 28 September 1945, a few months after the end of the last war in Europe. Thus his life spanned almost exactly the rise and fall of unified Germany.

'Aetherrausch'

In 1901, when Sudeck had just become Director of the Outpatients Department of the Eppendorf Hospital, he described his work on ether anaesthesia. He called his paper 'Das Operiren im ersten Aetherrausch', literally 'Operating in the first ether intoxication'.[3] In fact, he was making use of the analgesia which occurs during the first stage of anaesthesia. He described some 200 cases in which he used a simple ether mask, called a Czerny mask: a cylinder open above and below that contained stretched

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Fig. 1. Professor Paul Sudeck (Archives of the University Eppendorf Hospital, Hamburg).

flannel, on which he poured 30–50 ml ether, after the patient exhaled fully after several deep breaths; he then applied the mask to the face and asked the patient to breathe as rapidly and as deeply as possible, in order to distract his (her) attention from the start of the operation. The incision was made after the first or second deep inhalation of ether vapour. Such things as preparation of the skin, application of an Esmarch's bandage, were made before the ether inhalation began. The patient was urged to concentrate on ignoring the pungent smell of ether and it is hardly surprising that this helped to distract attention even further from the operation. For obvious reasons the patient was not allowed to enter the stage of excitement. Sudeck performed many minor procedures such as incisions of abscesses with this technique, but he also twice amputated legs in old and ill people without any complaints of pain and even opened and drained the abdominal cavity in cases of peritonitis, and was prepared to deepen anaesthesia if necessary. He did not claim the technique as a new discovery, but believed that it was too little used, since it produced virtually no complications. Vomiting was rare and the patients recovered all their faculties soon after the end of surgery.

Sudeck published another paper in 1902, describing his further experiences with this technique, again using the Czerny mask.[4] He recorded the different reactions of his patients, according to their temperaments, and stressed that consciousness and the sense of touch were preserved, but advised that, if the stage of excitement supervened, the

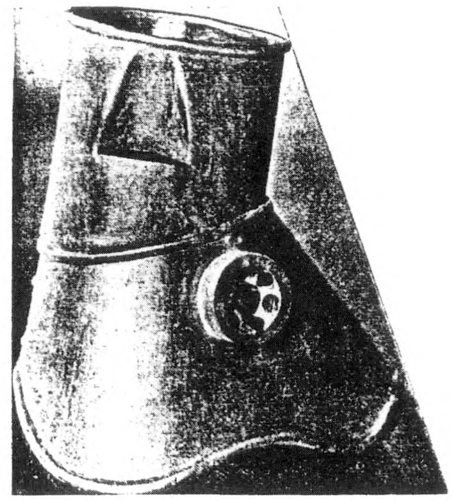

Fig. 2. Sudeck's original inhaler (by permission of G. Thieme Verlag, Stuttgart).

operation should be stopped and the patient allowed to recover; as the analgesia persisted for a short time, the operation could often be completed when the patient had regained consciousness. Sudeck obviously became expert in the method, and performed more major procedures, such as resection of lymph glands of the neck, curettage of bone, tendon repair and even resection of a small tumour of the median nerve and its resuture. Indeed, he stated that the management of surgical outpatients had led him to use only ether analgesia in operations which did not require complete stillness and muscular relaxation.

The Sudeck inhaler

Sudeck described his own mask in 1903,[5] which he used predominantly for ether, although he considered it suitable also for chloroform anaesthesia (Fig. 2). Made of metal, it consisted of a facemask taken from a contemporary Roth and Dräger chloroform and oxygen anaesthetic machine. The mask was moulded to the shape of the average adult face and seldom required a rubber rim. An upper chamber was attached to the mask, but was divided from it by a metal base in which was inserted a one-way inspiratory valve with a mica diaphragm. There was an expiratory valve of similar design on the side of the mask. In Sudeck's original mask, the upper chamber, as well as having an open top, had two small holes, one on each side; it contained gauze, on to which ether could be poured through the top in a supine patient, or through the side in either lateral position. The whole device was quite small, about 13 cm in height and 7 cm in diameter at the base of the upper chamber. It was therefore a nonrebreathing system with a deadspace of 80–100 ml in the mask portion only. It was easily sterilised by boiling except for the replaceable gauze. It was made by the Dräger factory and marketed by Leonhardt Schmidt and Co. in Hamburg and cost 12 marks (11s 7d) in 1903.[3,6] Later models were slightly bigger, with one large hole in the side of the upper chamber, which could be rotated according to the patient's position (Fig. 3).[7]

Sudeck used his mask for general anaesthesia, but also found it invaluable for the induction of ether analgesia. He used a premedication of morphine 10–20 mg, sometimes with up to 0.5 mg scopolamine half-an-hour beforehand,

Fig. 3. Sudeck's Inhaler, later model (by permission of the Association of Anaesthetists, Charles King Collection).

for longer operations. He stated in 1909[8] that he used a gentler form of induction, and allowed only a few drops of ether to fall on the gauze while the patient inhaled deeply and increased the drip-rate as the ether vapour was tolerated. He believed this method was particularly suitable for use by general practitioners who performed minor surgery.

Sudeck and nitrous oxide

In a paper published in 1926,[9] Sudeck compared the relative advantages of ether, nitrous oxide and Narcylen, the name given to purified acetylene. Narcylen was in common use in Germany at that time, and was much cheaper than nitrous oxide, which had to be imported from England. Acetylene, although highly explosive, had the advantage that it was nontoxic and could be used with 20% oxygen; however, it caused some muscle rigidity, was rather irritating to the airways and had a somewhat unpleasant smell. Sudeck gave a detailed and well-balanced account of the advantages and disadvantages of the three agents, and pointed out that, while ether could not be dispensed with at that time, especially for operations that required muscular relaxation, the greater potency of Narcylen and the higher oxygen concentrations which could be used compared with nitrous oxide should not be allowed to outweigh the dangers of explosion. He recommended that nitrous oxide should be used in spite of its expense and that its safe administration should be mastered.

In 1926, Sudeck and his assistant, Helmut Schmidt, devised an anaesthetic machine for use in thoracotomy, whereby the anaesthetic gases could be administered under positive pressure.[10] It was less cumbersome than Sauerbruch's pressure chamber, but depended on the mask's close application to the patient's face. The size of the

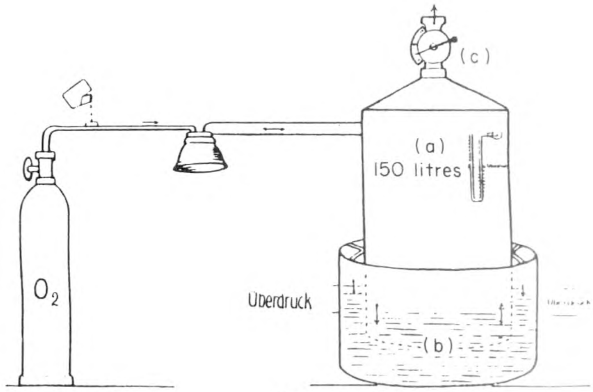

Abb. 3.

Fig. 4. Diagram of the Sudeck and Schmidt Positive Pressure Apparatus (by permission of Springer-Verlag, Heidelberg).

pressure-swing with respiration with the open pleura, and therefore of the 'pendelluft' or rebreathing from one lung to the other, and of the mediastinal 'flap', was reduced by using a spirometer with an adjustable pressure valve. The apparatus (Fig. 4) consisted basically of a spirometer and attached water manometer (a) with a capacity of 150 litres, dipping into a water bath (b). A nozzle (c) with a screw valve and pressure gauge regulated the outflow of excess gas and permitted an accurate adjustment of the pressure within the spirometer. A metal tube, 2.5 cm in diameter and 125 cm long, led from the spirometer to the mask, to which oxygen and anaesthetic vapour passed by a second delivery system. The anaesthetic, usually ether, dripped in by Venturi effect and siphonage from a reservoir. Up to 40 litres/minute of oxygen was allowed to flow into the apparatus, after the mask had been closely applied; the pressure within it was regulated at the nozzle (c). The flow was reduced to 4–8 litres/minute, which gave adequate oxygen and carbon dioxide exchange when the desired positive pressure, usually 1.0 kPa, was reached. The extent of the pressure swing was shown on the manometer since respiration was spontaneous; on inspiration the reduction in pressure was offset by an increase in the water level in the spirometer, while on expiration the level decreased and excess gas escaped through the nozzle (c). The bigger the spirometer and especially the greater the area of water exposed to the gas within it, the less the pressure swing; the best proportion was when the water surface area in the spirometer equalled that in contact with the outer air. The carbon dioxide content of the gases was low, even with a flow of only 2 litres/minute of oxygen, while the resistance to breathing was minimal. The design was simple although bulky, and it had been used for nitrous oxide and oxygen anaesthesia.

The 'Modell A' nitrous oxide anaesthetic machine

Sudeck and Schmidt had begun work in 1924 on an anaesthetic machine with a circle absorber system, using potash as the absorbent. It was manufactured by Drägerwerk AG and described in a manual dated October 1927 (Fig. 5).[11] Dräger claim that it was the first of its kind, since an experimental machine constructed for Franz Kuhn in 1906 had proved impractical, because of toxic decomposition by potash of the chloroform he employed. It consisted of cylinders of nitrous oxide (1) and oxygen (6) with pressure

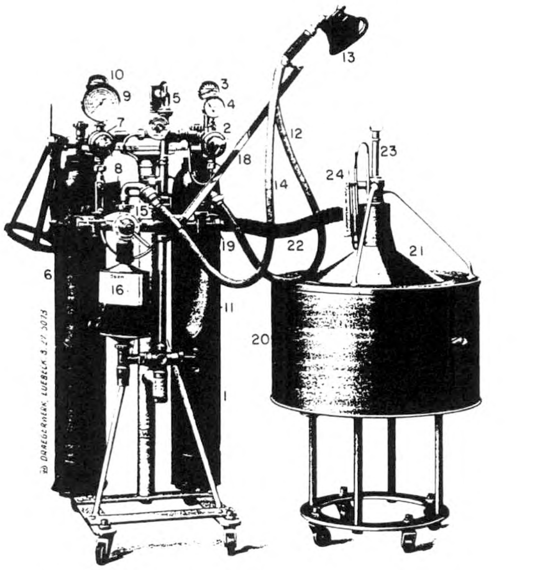

Fig. 5. The 'Modell A' Anaesthetic Machine with positive pressure attachment (by permission of Drägerwerk AG, Lübeck).

gauges (3,10) and pressure-reducing valves (2,7). Nitrous oxide flowed through a pressure-gauge meter (4); the flow was adjustable to 2, 4 or 6 litres/minute by means of a wing-nut, and then passed a drip-feed bottle (5), which could deliver ether at three rates between 25 and 200 drops a minute, to a mixing chamber (8), into which oxygen flowed through another pressure-gauge meter (9), permitting regulation to from 5% to 30% of the chosen nitrous oxide flow. The mixed gases passed to a reservoir bag (11) and thence via a wide-bore metal hose (12) to the facemask (13). The expired gases returned through a second hose (14) to a stopcock (15) at the carbon dioxide absorber (16) and back to the reservoir bag. The absorber, a canister that contained potash, could be partially or completely bypassed by turning the stopcock. Excess gases escaped through a tap (17), adjusted so that the reservoir bag was always full. A one-way valve at the bend in the pipe above the bag ensured that the flow of gases could be only in one direction. The flexible hoses were supported by a metal arm (18) to prevent pulling on the mask, on whose frame was a hole covered by a slide, which could be opened to admit air. The potash canister had a life of about 6 hours with continuous use; it was exhausted when it no longer warmed up, or rattled when shaken. Potash, being deliquescent, absorbed water from the gases. For thoracotomy, the positive pressure appliance was attached by a light flexible hose (22), after unscrewing the locking nut (19) on the side-arm above the reservoir bag. It consisted of a water chamber (20) and diving bell (21); the desired pressure was achieved within the system by depressing the push-rod (23) and was read off at the water manometer (24). The escape tap (17) was closed.

A gas-tight fit of the mask to the face was ensured by a head-harness attached to the frame of the mask. Premedication with morphine 10–20 mg and atropine 1 mg or scopolamine 0.5 mg was followed by induction with 100% nitrous oxide for 5–10 breaths, followed by the addition of oxygen 5%, then 10% and, after 20 minutes, 15–20%. A small amount of ether (usually 20–50 ml) was added if necessary.

The prime purpose of the machine was to reduce to a minimum the amount of ether and nitrous oxide required, although from 1927 the latter was manufactured at greatly reduced cost in Germany. The patient could often be maintained on nitrous oxide and oxygen alone where light anaesthesia was sufficient; the anaesthetist occasionally gave a few breaths of pure nitrous oxide 'provided there was no cyanosis'! The type of respiration rather than the cardiovascular state determined the level of anaesthesia and good results depended greatly on the expertise of the anaesthetist and his ability to recognise the early signs of hypoxia.

A model of this apparatus was said to be still in use at the University Clinic in Rostock as late as 1947.[12]

Conclusion

Sudeck was a pioneer in modern techniques of anaesthesia in Germany. His advocacy of ether analgesia for outpatient surgery and his practical use of nitrous oxide and oxygen in a circle system with carbon dioxide absorption, together with the option of applying continuous positive airway pressure in open chest surgery, make him worthy of a place in the history of anaesthesia.

Acknowledgments

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SPECIAL ARTICLE

The M1 air crash

The demands placed on anaesthetic and intensive care services of two hospitals

N. R. COAD, M. J. JONES, A. J. BYRNE AND M. L. PEPPERMAN

Summary

The M1 air crash provided an enormous challenge to the anaesthetic and intensive care services of the hospitals which admitted the survivors, many of whom had serious injuries. This account describes some of the problems which were encountered in two of the hospitals, details the workload imposed on the anaesthetists and the staff of the Intensive Therapy Units and identifies factors which, if improved, might advance the management of multiple casualties admitted from the scene of a major disaster.

Key words

Major disaster; anaesthesia, intensive therapy.

At 1826 hours on Sunday 8 January 1989, a British Midland Boeing 737-400 aircraft crashed into the western embankment of the M1 motorway at Kegworth, Leicestershire, only a few hundred yards from the runway of East Midlands Airport. This accident was unusual because of the large number of survivors, many of whom had multiple injuries. There were 88 initial survivors (of 126 passengers and crew); 86 were transported by ambulance or helicopter to one of three hospitals, and two survivors with minor injuries were taken to a fourth hospital. The response of the Accident and Emergency services has been described,[1] and details of the injuries have been published elsewhere.[2] This is an account of the problems encountered by the Departments of Anaesthesia and by the Intensive Therapy Units (ITU) at two of the hospitals involved, University Hospital Nottingham (UHN) and Leicester Royal Infirmary (LRI).

Distribution of casualties

Deficiencies in communications resulted in arrival of patients at each hospital in 'waves', with the result that each hospital in turn was almost overwhelmed with casualties. Most of the survivors transported initially were taken to Derbyshire Royal Infirmary, although those with a serious head injury were sent directly to UHN, which houses a neurosurgical unit. The total number of survivors admitted to each hospital is shown in Table 1. The first admission to UHN arrived at 2115 hours, and the last at 2253 hours. Leicester received its first patient at 2200 hours and the last at 0500 hours on 9 January; the majority were trapped in the fuselage and cut free from the wreckage.

Initial hospital management and disposal

Triage in the Accident and Emergency Department revealed that a large number of patients had sustained major injuries. The abbreviated injury scales[3] are shown in Table 1. One consultant anaesthetist in each centre remained in the Accident and Emergency Department, together with a variable number of junior anaesthetists who helped in resuscitation, and accompanied the most seriously ill patients during transfer to the operating department or ITU. Seven patients in Nottingham and one in Leicester required tracheal intubation in the Accident and Emergency Department.

Some patients were resuscitated at the scene of the accident by medical teams from all three centres; however,

Table 1. Total number of patients admitted, total number of injuries recorded and injury severity scores of survivors admitted to the three hospitals.

	DRI	UHN	LRI
Total number of casualties	25	40	21
Total number of injuries	94	140	90
Mean abbreviated injury score	12.1	11.5	14.5
Number of casualties with abbreviated injury score > 16	7	9	6
Number admitted to ITU	4	13	6 (4)*

DRI, Derbyshire Royal Infirmary; UHN, University Hospital Nottingham; LRI, Leicester Royal Infirmary; ITU, Intensive Therapy Unit.
*Four patients received intensive therapy in the theatre recovery area for some hours before and after surgery.

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many of the patients transferred initially received no resuscitation. There was no anaesthetist in any of the on-site medical teams, although two attended the scene fortuitously. Patients were assessed in the Accident and Emergency Department on arrival at hospital, but had to be cleared very rapidly to make room for incoming casualties. The nature of the injuries was such that there was little room for delay in resuscitation and little margin for diagnostic errors. 'Blanket' policies were adopted in Nottingham for fluid replacement, requirement for artificial ventilation and diagnostic tests. Documentation of injuries was often poor and this resulted in patients arriving at other sites in the hospital where the staff had little idea of their injuries or of the investigations performed. In addition, the rapid disposal of the less seriously ill patients led to problems in determining where individual patients had gone. The use of a purpose-built mobile ITU bed proved to be invaluable during transfer of critically ill patients within the hospital. A more selective approach during initial resuscitation was adopted in Leicester. Patients remained in the resuscitation room for a short time (mean 28 minutes, range 15–44). The 10 most seriously ill patients were moved after initial resuscitation and X ray to either the ITU or the theatre recovery area, which was used as an annexe to the ITU and was fully equipped for provision of cardiovascular monitoring and ventilatory support. There were difficulties in both centres in identification of cross-matched blood and laboratory results; initially, all requests were made using the patient's major incident number, but after the patients had been identified, requests for blood and biochemical or haematological laboratory results were often made using the patient's name.

The most common injuries were fractures of the lower limbs and (or) pelvis. Seven patients at UHN and seven at LRI had multiple rib fractures, and a total of eight of these required pleural drainage. Three patients at UHN and one at LRI had sustained a severe head injury. Three patients at UHN underwent laparotomy after positive abdominal paracentesis; one was found to have a ruptured spleen. One patient at LRI required laparotomy for repair of a ruptured bladder. Four patients in Nottingham and one in Leicester suffered a severe spinal injury.

Anaesthetic services

Four junior anaesthetists were available immediately in Nottingham (one senior registrar, two registrars and one senior house officer). They were joined rapidly by other anaesthetists, who arrived after they heard of the disaster from television news flashes or after a telephone call from an anaesthetist on the site; the hospital call-out procedure failed totally, and only the two consultants on call were summoned by the hospital telephonists. A total of 25 anaesthetists attended during the night. Three (one consultant) were based in the Accident and Emergency Department; two registrars were employed in transport of patients within the hospital and one consultant with ITU experience remained in the receiving ward to supervise resuscitation. Three consultants, two senior registrars, two registrars and three senior house officers were required for patient management in the ITU, and another consultant undertook administrative and organisational duties; it was found essential to allocate one doctor for each ITU patient during resuscitation.

Seven operating theatres were opened and used throughout the night; five consultants, one senior registrar and one registrar staffed the theatres, another registrar relieved these anaesthetists for breaks and another consultant was used to determine priorities and allocation of theatre space. Some of these anaesthetists were sent home during the night so that they could return later to relieve those who continued to work. Eleven patients underwent 38 separate operative procedures under general anaesthesia in the first 12 hours. Another 11 patients required 26 surgical procedures during the next 24 hours. Two operating theatres were used continuously for 48 hours. During the next 4 weeks, 30 operations were performed on patients from the accident.

Five junior anaesthetic staff were available immediately in Leicester (one senior registrar, three registrars and a senior house officer). They were joined initially by three consultants, two of whom had ITU experience; one took immediate responsibility for the ITU and one worked in the Accident and Emergency Department. Four more consultants were contacted when the scale of the disaster became apparent; one took charge of resuscitation in the theatre recovery area and the remainder worked in the operating theatres. Another anaesthetist acted as coordinator. Thirteen anaesthetists in all were involved in management of patients during the night. Ten patients required a total of 23 surgical procedures in the first 12 hours. One of these was undertaken in the Accident and Emergency Department and the remaining nine in the Operating Department, where two theatres were opened. Sixteen of the 21 casualties admitted to LRI required surgery. Anaesthesia was required for 23 secondary procedures in the 6 weeks after the accident. The total anaesthetic time was 68.5 hours (range 0.5–13).

Intensive therapy

In Leicester, there were three patients in ITU and six in the adjacent coronary care unit (CCU) at the time of the accident. The CCU was evacuated by transferring all patients to medical wards. One of the patients in ITU was transferred to a surgical ward, one to another ITU in the city and the third was considered too ill to be moved. Fortunately, there was a period of more than one hour between the accident and the arrival of the first casualty, and this lag period was used to organise the distribution of patients and staff and for preparation of facilities. A total of 18 'ITU' beds was made available (eight in ITU, six in CCU and four in the theatre recovery area). Five patients were admitted directly to ITU for resuscitation and stabilisation; three subsequently underwent surgery within 24 hours, one on the next day and one 3 days later. Five patients were admitted to the theatre recovery area for resuscitation and monitoring before surgery. One of these patients was transferred to ITU after surgery; the remaining four were discharged to the surgical wards within 18 hours after surgery. Two patients were discharged from ITU within 24 hours, three after 48 hours and the last after 6 days. One patient was readmitted for 2 days after major surgery 14 days after the crash. In all, 18 bed days were used in ITU.

In Nottingham, three of the nine beds in ITU were occupied. Two of the patients were transferred to other areas of the hospital and the third to an ITU at another

hospital in the city. Four additional ITU beds, which normally are not staffed, were opened. Thus, 13 ITU beds were made available for the casualties within 2 hours of the accident. Twelve of these beds were occupied in the first 12 hours. Eight patients were admitted directly from the Accident and Emergency Department, and all underwent surgery within 24 hours; the other four were admitted to ITU from the operating theatre. A 13th patient was admitted 24 hours after the accident when he developed fat embolism. The mean duration of admission was 10.6 (range 2–35) days; in total, 132 ITU bed days were used.

Mortality

One patient died shortly before arrival at LRI. In Nottingham, two patients died within 24 hours (one with severe head injury, one with a presumed ruptured thoracic aorta). One Nottingham patient died on the 14th day, from sepsis and multiple organ failure, and one on the 21st day, from pulmonary embolism. The TRISS[4] scoring system indicated that all of the patients who died in Nottingham had an expected mortality of more than 50%; however, four other patients with a similar expected mortality, survived.

Discussion

Fortuitously, this accident occurred close to three large hospitals, all of which were equipped to deal with a large influx of casualties. The number of patients with serious injuries would have overwhelmed any one hospital, and no single ITU could have coped with the need for 27 admissions. A number of lessons were learnt, although the major disaster plans in the three centres worked well in many ways.

Communication with those at the scene of the accident was poor. This resulted in transport of patients sequentially to each centre, with the result that each hospital in turn was presented with a large number of casualties over a very short period of time. In addition, each hospital was unaware of the number of casualties it should expect, and had no prior knowledge of the extent of their injuries. Poor communications have been reported previously during evacuation of casualties from the site of a major disaster, and may be improved by the incorporation of a doctor in the communication team.[5] A recent report[6] by the Royal College of Surgeons has drawn attention to the need for improvements in this area.

The degree of resuscitation at the site was variable, because initially it was decided to move survivors rapidly to hospital. A number of patients required management of the airway whenever they arrived in hospital. The presence of an anaesthetist at the site as part of each emergency medical team would have been valuable in management of airway problems, as well as in provision of general resuscitation. In West Germany, considered by some[7] to have one of the best accident services in the world, only doctors with experience of intensive care are included in emergency medical teams.

The deployment of anaesthetists in the Accident and Emergency Department proved to be invaluable in provision of appropriate fluids, analgesia and sedation, and ensured that the severely ill patients were accompanied by suitably experienced personnel during transfer for investigations or definitive therapy. A fully equipped mobile ITU was also valuable. There was little time for resuscitation in the Accident and Emergency Department. General rather than specific criteria were used for treatment and diagnosis in Nottingham; the adoption of simple algorithms for resuscitation was shown to save time, reduce errors and decrease mortality in similar situations.[8,9] However, there may be a risk that treatment is given which is not strictly necessary and, if time allows, the more specific approach adopted in Leicester may have advantages. Patients who require immediate surgery should be moved directly to the operating theatre suite for resuscitation,[10] and this occurred in both hospitals. Poor documentation could have been improved by the completion of an injury checklist before the patient left the Accident and Emergency Department.

In Nottingham, the need for rapid disposal of the very large number of casualties from the Accident and Emergency Department resulted in difficulty in determining their destination subsequently, and uncertainty in the operating theatres and ITU about the number and location of patients who might require treatment in these areas. The incorporation in the case notes of tear-off slips which are sent to the coordinating office whenever a patient is moved to a new destination (a system used by the army) would have been helpful. The most seriously ill patients were accompanied by an anaesthetist during intrahospital transport; this enabled continuity of care and eased communication problems about the patient's injuries and investigations.

Problems were encountered in both centres, because of the use of identification numbers, and later names, in relation to blood transfusion, blood products and laboratory results. These difficulties would have been avoided if the incident number had been used at all times.

The hospital call-out procedure failed, and the hospital switchboards were overwhelmed. In Nottingham, the existence of a direct telephone line in the Department of Anaesthesia was critical. The deployment of anaesthetists as coordinators in the resuscitation areas, operating theatres and ITU proved to be extremely useful to ensure that the requirement for anaesthetic resources was predicted and fulfilled. It was clear that anaesthetists were required not only for the few hours after the accident; a second 'team' of anaesthetists was required after the first 12 hours to relieve those who had worked all night. This was achieved by asking some anaesthetists who offered help, initially to remain at home, and by sending some anaesthetists away during the night after there was no longer a need for their presence.

It is inevitable that after any major accident there are numerous enquiries from relatives and from the media. Initially, it was almost impossible for relatives to obtain information by telephone, and it would be desirable for the ITU to have had a direct telephone line. It was found useful, after the initial phase, to give information to one nominated relative for each patient, and to make that person responsible for relaying this information to the other relatives; this saved time and possible confusion. The media have a valuable role in helping to disseminate information. It was unfortunate that, despite a regular update, they published some stories which misled and distressed relatives.

It was soon apparent that a disaster of this size creates

difficulties for several weeks. Elective surgery was abandoned for 24–48 hours, but there was a continued need for trauma operating sessions for a much longer period, and this necessitated cancellation of a number of elective operating lists. The ITU in Nottingham remained extremely busy for 3 weeks, and this resulted in the postponement of elective surgery for several patients who required intensive monitoring or support in the postoperative period. These longer term problems also must be considered when planning provision of services after a major disaster.

Acknowledgments

We acknowledge the help of all the anaesthetists who were involved in treatment of the casualties from the aircraft. This account concentrates on the role of the anaesthetic and intensive care services, but the authors acknowledge that the anaesthetists involved were members of much larger teams of medical and nonmedical staff who dealt with the patients.

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SPECIAL ARTICLE

Clinical and resource management: what are the options for anaesthetists?*

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The choices in clinical and resource management offer a significant challenge to anaesthetists in the 1990s and this challenge is a very appropriate theme for a Pinkerton lecture. Dr 'Tony' Pinkerton's career reveals him to have been a man well qualified to have met these challenges which now face the profession. In addition to a distinguished clinical career in a department that became a centre of excellence in teaching at the Western Infirmary in Glasgow he occupied the post of consultant-in-charge of the anaesthetic service for 12 years and history records him as an eminent administrator and manager. One has the sense on reading his obituaries of a man who would have taken departmental and resource management in his stride without any diminution in the quality of clinical service or of the responsibility of doctors to shape and direct the clinical services to patients. It is a pleasure to have the opportunity to make this Pinkerton lecture a tribute to his career.

It is necessary to define the terms clinical management and resource management with more care before the detail of this argument is outlined. The term clinical management embraces three aspects in the day-to-day work of most admitting consultants. The first is the clinical management of the individual patient, the second is the management of waiting lists, both inpatient and outpatient and the third, and increasingly more complex, is the management of the clinical team or network that focuses upon the care of the individual patient, his diagnosis and treatment: a management task that carries with it the responsibility to ensure that all the necessary and appropriate clinical skills are used in diagnosis and that the subsequent treatment is consistent with that diagnosis. It is important to recognise that at every stage of that work patients and their treatment are being managed, and in that sense consultants are therefore, managers.

Resource Management has confusingly come to have both a wider and a narrower definition in the context of our current changes. The wider definition of resource management encompasses the total task of organising buildings, equipment, staff, supplies and financial resources in such a way as to enable clinical managers to treat their patients appropriately.

The narrower definition relates to the current resource management initiative (RMI) which has become yet another NHS acronym. The phrases clinical budgeting, management budgeting and resource management have been used in succession since the first Griffiths report in October 1983.[1] They are used to define a process by which doctors and nurses become involved in the management of clinical services, the measurement of clinical outcomes and the identification and measurement of clinical and financial inputs as part of the process of directing that clinical service. This brief definition does not do justice to all the current variants of RMI but it does define the key principles involved.

The broad task of resource management was ultimately determined and controlled by hospital consultants before the late 1970s. During this period the NHS operated a cost-plus system of funding health care. Health authorities, whatever their title, were given the previous year's money together with development money and those developments were the ones shaped and determined by hospital consultants, either because they were additional appointments or as the result of service developments by the existing team of consultants. It was the job of hospital administrators and treasurers to ensure that the equipment was bought, the staff were employed and, if necessary, the buildings were put up; but the decision about what to develop rested with those who were the clinical leaders of their local services. Resource management was in this sense indeed directed and controlled by consultants and no hospital administrator or treasurer would dream of saying, for example, that a consultant could not see more people with a particular condition or could not use a particular drug.

The crucial change, the consequences of which are still being worked through, was the recognition in the late 1970s that national resources were simply inadequate to fund every new development. In 1976 the International Monetary Fund warned the Government of approaching insolvency and a condition of its loans to bale out this country was a reduction in runaway public spending that was growing beyond the ability of the nation to fund it. The following year saw the publication of the Resource Allocation Working Party Report[2] which initiated the redistribution of funds between regions on a weighted population basis, with standardised mortality ratios used as a proxy for morbidity. It quickly became apparent from then on that consultants could not simply expand their services and expect resources to follow. Consultants had increasingly to seek support from the nonmedical administrators of the past who became the general managers and finance directors of resource management. For the first time in the history of the NHS they became able to exercise a significant degree of control over which clinical developments took place and which did not.

Ever since these changes became apparent the medical profession has been faced with a clear choice, either to become involved in the management structure, as the controllers of resources and budgets and as directors of their clinical services, or to stand back and see these tasks falling to nonclinical managers or other clinicians such as nurse managers. Where consultants opt out they must accept that these others will have to take the final decisions about resources and will have to arbitrate between consultants. The relationships between consultants and other health care staff will change as a consequence and however

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slowly, changes in training, numbers, and deployment will follow.

The catalyst for the organisational changes that we are now going through is recognised world wide as the ability of medical science with its continual development and increasing sophistication to outstrip growth in gross domestic product (GDP). The health care system is merely adjusting to live with the consequences of that.

There is clearly a choice for consultants to participate or not in the new RMI. Six pilot sites are now to be expanded and next year there will be 50 more. The British Medical Association (BMA) is at present concerned that these first sites have not been evaluated, but without debating that, there is a prior issue of principle involved. A key aspect of resource management is to identify the cost of treating a particular case. Variation might occur genuinely on grounds of efficiency or because clinical management differs between hospitals or consultants. Your senior colleagues have often been wary of supporting this resource management initiative, because they fear that the cheapest clinical option will be imposed, and so damage prospects for some patients. Those who see RMI in this light regard the initiative negatively.

The alternative case is based upon the method of funding clinical treatment and upon a desire to protect clinical services in the future. The traditional method of funding a clinical service has been to allocate fixed resources annually and to expect that somehow everything will be done within that resource. Arguments that resources are inadequate to meet the needs of patients cannot be formally proved and are met with unspecific requests to improve efficiency. RMI offers a way out by enabling consultants to identify properly the costs per case by clinical type and hence relate the volume of clinical service to resources. General managers, health authorities and even Secretaries of State can be told the resource implications of requests for clinical service, and the comparison of costs between consultants and hospitals is essential to demonstrate the financial efficiency of a service.

This does not ease the anxiety of consultants who fear that they will be forced to accept only the cheapest choices. It is at this point that medical audit is crucial to the debate. The Colleges and this Association are heavily involved in the development of systems of medical audit that are concerned with clinical alternatives and clinical outcomes. It is clear that the White Paper[3] initiatives will lead to the introduction of medical audit and the Colleges are intimating that recognition for training will depend upon an effective operating audit system. This audit system is vital to protect the patient and clinical practice in an age of resource management. If, and when, audit identifies the more successful and appropriate clinical procedures, resource management will identify their legitimate cost and this in turn will identify the budget required for the service. Where money is not forthcoming under this system the extra volume of clinical service cannot be provided. Some consultants now see this as an essential safeguard for the future. Whichever view consultants adopt their choice is crucial for the way in which they will practise in the 1990s.

The final area of choice concerns clinical management and in particular the management of the multidisciplinary clinical team or network that focuses upon the care of the individual patient. As that team has grown in the last 30 years, so the task of ensuring that diagnosis is followed by appropriate and consistent treatment has become more difficult. At the start of the NHS consultants (specialists) were seen as exercising overall clinical responsibility for hospital patients; as a result they exercised a leadership role within the multiprofessional team of doctors, nurses, radiographers, technicians, physiotherapists etc. This leadership concept is now coming increasingly under challenge as a model for decision-making. Examples of consensus and democratic decision-making teams exist in some specialties but, more generally, there is a growth of anarchy. Anarchy in the sense that individual professions are determining their own way of treating or caring for patients without that method of working being necessarily consistent with the treatment given by others, or even with the medical diagnosis. Those who dislike the use of the word anarchy prefer to describe this as cross-referral between professional colleagues who each have the responsibility to do what they see as best whilst the patient is in their immediate care. This view of 'collegiate' responsibility is at odds with the traditional concept of the overall clinical responsibility of consultants.

We are now witnessing the development of a clinical hierarchy in nursing in place of the older and diminishing management hierarchy which involves clinical nursing specialists with authority over ward sisters. This is combined with the concept of primary nursing on the wards where, for example, a staff nurse takes responsibility for a group of 'named' patients for whom she institutes and carries out care plans; other nurses are the junior members of her team. The clinical nursing specialists are seen equally as developing 24-hour responsibility for their service and include on-call commitments. This copying of medical hierarchies and responsibilities carries implications for medical access to patients and, in some hospitals, declining support and attendance on the ward rounds conducted by doctors.

Examples are legion and cannot all be listed here, but whether anaesthetists meet this with midwives in obstetrics or nurses in ITUs and theatres they are developments that cannot be ignored. These developments pose choices and the medical profession has not yet displayed any consistent response.

The consultants of the 1990s will have to decide whether and to what extent they resist overall clinical responsibility eroding gradually into a collegiate relationship of near equals, described above as potential anarchy. If the question is avoided the erosion is inevitable and if different consultants react differently in a hospital with no common purpose or policy the change will be painful in terms of working relationships with other professionals: as any anaesthetist knows who has tried to run an ITU with a different policy for every surgeon who uses it. The fact that the leadership model is in the patient's interest does not of itself ensure that this model of decision making will remain.

The government's White Paper, 'Working for Patients',[3] linked to the new RMI is bound to become a focus for the changes and options faced by doctors in the next 5 years. It stresses the importance of doctors in management and ties the medical audit system in part to management involvement. It encompasses medical audit and offers sanctions against those who fail to participate. It offers money to hospitals that accept the RMI. It hints at the ending of the Pay Review Bodies, certainly for doctors newly employed in self-governing hospitals. For all these reasons those who regret the necessity for change can blame the White Paper and identify it as the cause of all the unpleasant choices ahead. The BMA's rejection of the White Paper will make this focus of blame easier.

In reality, however, the White Paper is only the consequence of a much more fundamental cause of change. Once it is recognised that the growth in GDP cannot keep pace with the advance in clinical science some more substantial form of resource control is necessary to balance supply and demand. So far, whether the examples are taken from the United States of America, West Germany or France, the types of controls are largely the same: to squeeze more health care per unit of currency and to encourage families

to become more responsible for postoperative care. The present White Paper is also a supply-side initiative, and shares similar methodologies with each of these other countries, and many of its initiatives actually predated January 1989. Decentralisation of management and the encouragement of doctors to play a more active role in management began with the implementation of the first Griffiths Report in 1984. Clinical budgeting experiments (now resource management) began the year after in 1985. The medical profession has been leading on medical audit for the last 2 years. Those doctors who have wished to resist or ignore these changes over the last 5 years will find it easier to focus opposition on the White Paper, but they will be opposing a symptom rather than the real cause of change. Our present Government had to act just as the Labour Government of 1977 had to act on the Resource Allocation Working Party Report and for the same basic reason, that developments in medical science and the spending opportunities they create are outstripping the growth of GDP.

Finally, there is a sense in which all these choices for the future are linked. If consultants hold the budget and direct the clinical service they will reinforce within the management structure the continuation of the leadership model in the diagnosis and treatment of patients. Conversely, if the nursing profession comes to dominate the roles of budget holder and clinical service manager their view of a collegiate clinical relationship with medicine will develop more rapidly. If consultants become involved in the resource management initiative and medical audit, both their management role and their clinical leadership will be reinforced. If they do not, the control and direction of resource management will increasingly fall to others and the White Paper indicates that general managers will call in outside consultants to perform 'independent' medical audit.

In summary, what this leads to is, that the consultant who says, '... I just want to stay as I am practising as I always have done with no management tasks and computers to distract me', cannot have that wish in the future. It is not one of the options, simply because an individual who keeps his or her head down cannot compel everyone and everything around to remain unchanged. The current extent of change and turbulence in the NHS is also far too much to hope that things will stay unchanged by accident. Consultants of the 1990s have the opportunity to lead the system for the future in the patients' interest but only if they get involved and are prepared to meet and overcome the opposition they will inevitably encounter. From the evidence of his career there seems no doubt how 'Tony' Pinkerton would have responded, it is now for you to take up the challenge.

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SPECIAL ARTICLE

Anaesthesia and the law

Respiratory failure in a young child after aspiration of hot tea

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At 1745 hours on 18 July 1980, when Zeena Sa'd was nearly 19 months old, she sucked near-boiling tea into her mouth from the teapot spout. Her mother and aunt rinsed out her mouth with cold water and then the mother telephoned their general practitioner, (GP) Dr R. He examined the child at the surgery a few minutes later. The mother explained what had happened. The doctor told her not to worry. He opened the child's mouth and checked inside; she had a scalded lip, was spitting and saliva was continuously coming from her mouth. The doctor prescribed Calpol and Asilone and said the child would be 'Okay'. The prescription was quickly dispensed by a local chemist and the child taken home, still crying.

The child was still crying and in great pain and her saliva was still flowing freely at 2000 hours. The mother telephoned the surgery again but was referred to Dr R's partner, Dr D. The mother once again explained what had happened. Dr D. said that it would take about 2 hours for the child's condition to improve, but that she should telephone again if she were troubled.

After about 10 minutes, the child's condition seemed to improve though she was still dribbling. However, just before 2200 hours the child began to heave and to cry very severely; the mother became very worried and again telephoned Dr D. who arrived within 10 minutes. He examined the child, checking respiration, pulse and mouth, and asserted that although there was no cause for alarm she would be better in an environment where she could be observed. He suggested that she be taken to Epsom District Hospital. Having established the parents knew the way to Epsom, he gave directions with the aid of a sketch map. He telephoned the casualty department to warn them of the child's arrival explaining that she had drunk something hot a few hours back and that this could lead to respiratory problems and that she would be better kept in hospital. Before leaving, the mother and nanny changed the child's clothes as they were wet with mucus and saliva. No urgency was suggested by Dr D. who apparently did not anticipate that the child's condition would deteriorate *en route*.

During the half-hour journey to the hospital, the child's breathing became increasingly laboured and her gasps for air assumed a higher and higher note. On arrival, the mother explained that it was an emergency, but unfortunately the parents went to the children's ward. The SHO paediatrician recorded *inter alia*, '10.45 pm. On admission, very distressed, pale with loud inspiratory stridor and sternal indrawing . . . acute laryngeal obstruction secondary to drinking hot tea'. He telephoned the consultant paediatrician, Dr Ransley who advised him to give 100 mg hydrocortisone and 100% oxygen with humidification and to attempt to insert an intravenous line, but the latter did not prove possible. The SHO anaesthetist was called but was unable to play a useful part.

About 12 minutes after the telephone call, the paediatrician arrived and at once inserted the intravenous line, but the obstruction was becoming more complete on account of the mucus. The child then suffered an anoxic fit which lasted for a minute or two; thereafter while the child was still unconscious, Dr Ransley, with some considerable difficulty, was able to insert a small tracheal tube. By then, as the notes indicated, apnoea may have subsisted for an indeterminate period. Gradually, the child became more conscious, and after a dose of valium, she was transferred to a specialist hospital accompanied by her mother and Dr Ransley, but by this time the child had suffered serious irreparable brain damage.

Negligent failure to admit child promptly to hospital

A claim in negligence was brought against the child's mother (dropped before trial), the two GPs, the paediatrician and the local hospital authority. Leggatt J. exonerated the paediatrician and the hospital authority but found the two GPs negligent. He said they had both been told that the child had *sucked* hot tea from the spout of a tea pot, but neither of them at the time had recognised the essential difference between 'ingesting tea from a spout and drinking from a cup. They should have done so. The difference is crucial, because sucking it from a pot may involve the inhalation of steam and may in any event mean that the liquid will be close to boiling point. It may also allow the tea to reach the throat directly without burning the mouth.' There had been no injury to the mouth such as would have been likely if the child had drunk from a cup.

At trial, Dr R. had acknowledged that the risk of injury to the stomach which he recognised, entailed also the risk of injury to intervening parts of the throat, such as the larynx. He accepted, as did most of the medical experts, that if he had realised the child had *sucked tea from the spout* he should have referred her to hospital, and in failing to refer her he had been negligent. It would not have been sufficient for him merely to have warned the mother to get in touch with him if the child's condition deteriorated, nor did he do so.

However, the judge said he felt sympathy for Dr R., since it was apparent that he would have been saved from the consequences of his breach of duty had his partner, Dr D., not also been negligent in circumstances which were undoubtedly more blameworthy. He should have visited following the mother's telephone call at 2000 hours; in failing to visit and to refer her to hospital he was negligent. Up to that stage if she had been referred to hospital she would not have suffered any permanent injury. When he did visit at 2200 hours admission to hospital had become more urgent than when she was seen by Dr R., and with a history of respiratory difficulty, her clothes wet from mucus or saliva, Dr D. failed to realise that her condition required urgent hospital admission. Some respiratory difficulty probably should and could have been detected with a stethoscope. The urgency was such that he should have shown the parents the way himself. He could then have ensured that the child went to the casualty department rather than the paediatric ward. As it was, he had failed to

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tell the parents that she should be taken to the casualty department.

The children's ward is the wrong place in an emergency

When he had telephoned the hospital, Dr D. had negligently failed to explain the seriousness of the child's condition. Had he done so, a team consisting of the consultant paediatrician and her SHO, the SHO anaesthetist, together with the casualty officer himself, would have been assembled to meet her and the hospital would have ensured that she went to the casualty department.

The judge said that 'If there had been a team ready to receive Zeena, then, despite the paediatrician's reluctance to intubate, I find her condition would not have been allowed to deteriorate so far as to become anoxic and that she would probably not have suffered brain damage.'

The judge exonerated the paediatrician. All the experts had agreed that the circumstances were very unusual if not unique. None of them had ever encountered thermal injury of this kind to the throat of a young child. Their researches had revealed only two examples of such injury in medical literature. In those circumstances, knowing now, that the child suffered from anoxia and had a fit and convulsion, there was a risk of wisdom with hindsight.

Some difference of expert opinion

Seven consultants were called: five anaesthetists and two paediatricians and there was also a GP. There was disagreement—some experts considered that the primary need was to relieve the obstruction of the child's airway by immediate intubation, but others thought the first duty was to assess the child's condition. Oxygenation was a prime requirement but this was put in hand. There were mixed opinions as to whether an intravenous line was desirable, irrelevant, unnecessary or a time wasting measure. In any event, there was no evidence that the child's condition was worsened by it. Administration of hydrocortisone plainly had no immediate effect but in the longer term would have helped to contain and then reduce the swelling. With regard to intubation the experts differed. At one extreme the view had been expressed that if there is obstruction or near obstruction then an anaesthetic is a nicety to be dispensed with, at the other end of the spectrum was the view that a general anaesthetic was the only safe way to intubate a child.

The situation confronting the paediatrician had been unique: whether to summon a consultant anaesthetist was within her discretion, and in not doing so before or in the course of the cascade of events in which she was embroiled, there was no failure on her part to exercise the ordinary skill of a consultant paediatrician.

Logistics and the need for teamwork in emergencies

The most cogent criticism of the paediatrician was that she had failed to arrange the attendance of a consultant anaesthetist (who could have intubated more quickly), but the judge found that the paediatrician's real problem was that the child was in the wrong place and that summoning a consultant anaesthetist would have made no difference to the outcome. There was no evidence of how long it would have taken the three consultant anaesthetists who lived further away than the paediatrician to reach the hospital. Further, if a general anaesthetic was to be administered to the child, she would have had to be moved to the operating theatre or to the casualty department both of which were some distance away. That process would have put the child at risk of precipitating acute obstruction and would also have taken time. The alternative would have been to move the anaesthetic equipment to the ward and that too would have taken time. By the time either of these courses had been adopted it would have been too late. Thus, even if the paediatrician should have sent for a consultant anaesthetic it would have made no difference to the outcome.

Dr D. had led the parents to believe that they should go to the children's ward by saying that the child would be better in an environment where she could be observed. That plainly contemplated an overnight stay. There had been no message left with or for the casualty officer by the GP as should have led him or anybody else at the hospital to assemble a team for the child's arrival. The hospital authority was accordingly not vicariously negligent either in this matter or in its treatment of the child.

The total sum awarded by way of damages against the two general practitioners (who were jointly represented) was agreed in the sum of £535,000.

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Correspondence

Is halothane obsolete? Two standards of judgement

Professor Weis and Dr Engelhardt (*Anaesthesia* 1989; **44:** 97–100) have again[1] given their view that double standards are applied in the criticism of various drugs and techniques in anaesthesia. Their argument is based, first on the risks from halothane being less, and sometimes considerably less, than those of other potentially lethal drugs or techniques. Second, because the animal models do not exactly conform to the human scenario, animal data are of little relevance. Third, since the tests used to establish the diagnosis of halothane-associated hepatitis are positive in less than 100% of patients (believed on clinical grounds to be so afflicted), then the tests have little validity, and finally, as the alternative agents may also cause hepatitis (although this may be an event of even greater rarity) the advantage of those alternatives must be viewed with scepticism.

Halothane-associated hepatitis continues to cause morbidity and, very rarely, mortality despite warnings against its use in specific patients known to be peculiarly at risk.[2] Surely the tragedy of even a solitary avoidable anaesthesia-related death is not diminished simply because its cause is not fully established or is of extreme rarity? The Confidential Enquiry into Peri-operative Deaths[3] (CEPOD) has established that the contemporary risk of avoidable death attributable to anaesthesia alone is of the order of 1 in 185 000. To be fair, none of the 4034 deaths included in the CEPOD report was attributed to hepatitis associated with halothane.

The evidence[4–7] for the existence of fulminant halothane-associated hepatitis based on antibody studies should not be overlooked. Similarly, the frequent occurrence of transient, and seemingly clinically insignificant, elevation of liver function enzymes after halothane cannot be denied,[8–11] especially when this appears to occur less readily after the alternative volatile agents.[12–13] The incidence of liver dysfunction is considerably higher in some groups of patients: for instance, Zaric and colleagues[10] found that approximately one in 300 postoperative patients (four cases) aged 45 years or older, with otherwise unexplained nausea, vomiting or pyrexia, had altered liver function tests. Two of these four cases developed severe liver failure.

As Weis and Engelhardt have already stressed,[1] the risks of a particular anaesthetic cannot be considered apart from the overall risks of anaesthesia. Currently there are fewer fatal cases in Britain each year of maternal aspiration of

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gastric contents than fatalities from presumed halothane-associated hepatitis and yet the vigorous precautions taken in obstetric anaesthesia would not have become widespread without the publicity given to the problem. Likewise the occurrence of malignant hyperpyrexia is of a comparable order to halothane-associated hepatitis and yet normal precautions against it occurring, even in family members of a susceptible patient, would include total avoidance of triggering agents.

Professor Weis and Dr Engelhardt answer the original question of whether halothane is obsolete only by implication, and indicate some of the potential disadvantages of the alternatives, enflurane and isoflurane. The time has now come for anaesthetists to make an individual choice, based on the particular circumstances of each patient and not on whichever vaporizer is presented. The production of surgical operating conditions by the use of powerful anaesthetic drugs to obtund responses inevitably will engender risk. The solitary standard of judgement is the decision based on knowledge of the advantages and disadvantages of the alternatives, and as such halothane is not obsolete, but being judged.

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A reply

We were not surprised to read an encore[1] of Dr Colin Blogg's view on the further use of halothane. In a personal letter (16 March 1988) Dr Blogg already confirmed our point: 'I have to agree with you that halothane is judged by a standard different to other anaesthetic agents or techniques, but surely this does not excuse repeated use of halothane when there is plainly a previous adverse reaction or a suitable alternative for that patient? ... I would certainly not press for the abolition of halothane in the present circumstances.'

We do not question the existence of fulminant hepatitis after halothane. We wonder, however, why Dr Blogg does not cite a more recent publication which proves that enflurane metabolism produces covalently bound liver adducts recognised by antibodies from patients with halothane hepatitis?[2] This study very clearly suggests that enflurane might be potentially immunogenic and thereby induce hepatic necrosis. New statistics are not available on this topic and although this study does not (yet?) incriminate isoflurane, we anticipate arguments against these substances too. Anaesthetists should be very reluctant to discard halothane, one of their best known and studied anaesthetics.

Our argument is for a rational, rather than emotional, judgement of halothane. We therefore stressed present contraindications to halothane and this certainly includes that the reliable anaesthetist must make an effort to detect the individual patient at risk and must be prepared to provide 'halothane-free' anaesthesia.

Given these precautions we hold to our view that anaesthesia will not become more safe by the abandonment of halothane in favour of its alternatives. On the contrary, we repeatedly see anaesthetists who have given narcotic, intravenous, anaesthesia without adequate postanaesthetic care in medicolegal cases.[3]

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The management of hyponatraemia

Hyponatraemia after transurethral resection of the prostate (TURP) and ultrasonic vesical lithotripsy has recently been the subject of discussion.[1,2] Absorption of irrigation fluid from the prostatic bed leads to the TURP syndrome which manifests itself as restlessness, confusional states and seizures from cerebral oedema as well as acute heart failure,

arrhythmias and pulmonary oedema.[3] This can occur within 15 minutes from the onset of irrigation.[4]

The syndrome is caused by water intoxication and is seen as the biochemical abnormality of dilutional hyponatraemia. Serum sodium levels of 120 mmol/litre or less are associated with significant morbidity and mortality.[5]

The treatment of acute water intoxication is controversial. It has been suggested that a rapid correction of the abnormality can itself lead to serious neurological damage which is manifest by central pontine myelosis (CPM).[6] Arieff[7] reviewed the published cases of CPM and concluded that it was more likely to occur in malnourished alcoholic patients and was not related to the speed of correction of the hyponatraemia. Early diagnosis and treatment are most effective in reducing morbidity and mortality. He suggested that hypertonic saline should be infused at 70 mmol/hour which will raise the serum sodium by 2–3 mmol/hour. However, since hyponatraemic seizures can cause permanent cerebral damage, the initial corrective phase may have to be more rapid.

The reduction in cerebral oedema used to be treated with mannitol[8] but it is well known that mannitol can itself cause a rebound increase in intracranial pressure which may cause further insult to the brain.

The problem of rapid treatment of water intoxication was tackled by Worthley *et al.*[9] They recommended the use of 29.2% intravenous saline 50 ml to be infused over 10 minutes for the treatment of this condition. This concentration was chosen since it approaches saturation and allows a large amount of saline to be given in a small volume. This raised the serum sodium by 3 mmol/litre within 30 minutes and successfully treated the hyponatraemic-related seizures without complications. Repeat aliquots were given as necessary.

Saline 29.2% is not commercially available for use in the United Kingdom. However, in the preparation of solutions for total parenteral nutrition by hospital pharmacies a saturated solution of saline is prepared. It is therefore readily available from local hospital pharmacies and is cheap (a 20-ml ampoule costs £1.70).

We would be interested to hear other views on this method of treatment. The TURP syndrome is underdiagnosed and there are no comprehensive guidelines for its treatment. We recommend that 29.2% saline is made available for its treatment.

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Apnoea after retrobulbar block

In their article (*Anaesthesia* 1989; **44:** 26–7) Drs Rigg and James raise some points that can be further developed.

First evidence of central spread of local anaesthetics from the orbit can vary from mild confusion, through convulsant behaviour, bilateral brainstem nerve palsies (including motor nerve blocking to the contralateral orbit with amaurosis) with or without loss of consciousness, to apnoea with marked cardiovascular instability.[1-6] The degree of development of the syndrome is dependent on the amount of drug which gains access to the cerebrospinal fluid. Small amounts cause minor sequelae whereas, with larger amounts, the fullblown picture of loss of consciousness, complete deafferentiation, apnoea, flaccid paralysis of all motor groups and hypotension with bradycardia from sympathetic blockade in the spinal cord may occur.[2] Intermediate amounts may result in the interesting clinical picture described below. In the writer's reported experience with 12 000 patients[3] onset has always been within 8 minutes of orbital injection; hence the patient should not be draped for surgery until 15 minutes have elapsed. Pulse oximetry is valuable and desirable.[3]

Drs Rigg and James state that instances of severe cardiovascular collapse are not being reported. They cite a paper by Rosenblatt, May and Barsoumian in which cardiac arrest was reported. In another article 'cardiopulmonary resuscitation was sustained for a period of 45 min before the patient recovered.'[4] They comment on the possible irritant effect of tracheal intubation. Fourteen episodes of central spread in the writer's practice were managed safely without intubation; this avoids problems when a patient recovers consciousness with a tracheal tube in place.[3]

In their first case, the occurrence of arterial hypertension and tachycardia was probably related to bupivacaine reaching the brainstem (bilateral vagal and glossopharyngeal nerve blockade cause simultaneous absence of vagal cardiac inhibition and interruption of the carotid sinus reflex),[2] yet insufficient drug was present to descend to the spinal cord. Marked increases in arterial blood pressure and heart rate after retrobulbar blockade have been reported.[2-5]

In their second case, Drs Rigg and James report bradycardia which probably indicated spread of the local anaesthetic drug at least as far as the thoracic spinal cord with sympathetic denervation of the heart. This has usually been accompanied by arterial hypotension[1,2] (Drs Rigg and James do not comment on the initial arterial pressure). The picture is very similar to that of 'total spinal' from excessive dosage of local anaesthetics by the lumbar route or inadvertent lumbar 'epidural' injection into the subarachnoid space. Positioning and circulatory support with vagolytics and vasopressors are important in clinical management.

Contrary to the statement in Drs Rigg and James' report, the importance of needle length used in orbital blocks is stressed.[1,6] To avoid cerebral spread the needle tip should not extend more than 3 cm beyond the orbital rim, nor should it be placed more medial than the sagittal plane

of the lateral limbus. During performance of the block patients should be instructed to direct their eyes in the primary gaze position rather than the old teaching of upward gaze with adduction, which brings the optic nerve closer to the needle tip.[1,5,6] Malposition should be suspected if there is resistance to injection.[4]

Peribulbar techniques may, as an alternative to retrobulbar block, be less prone to sequelae of the above type, although onset of akinesia will be somewhat delayed.[3] In addition, having anaesthetists perform orbital blocks may be safer for the patient because of closer monitoring.[2,5]

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A reply

We thank Dr Hamilton for drawing our attention to articles which discuss the hazards of long needles, and the risks of the patient looking medially and superiorly during needle insertion.

Dr Hamilton discusses the possible causes of cardiovascular changes seen during our cases of apnoea. It is probable in our first case that brainstem blockade coupled with the stimulatory effects of tracheal intubation were the causes of hypertension and tachycardia. However, in our second case it seems unlikely that local anaesthetic spread as far as the thoracic spinal cord. Apnoea only lasted 5 minutes and initial bradycardia and hypertension were reversed by positive pressure ventilation alone. This time sequence and easy reversibility would not be expected with extensive spinal blockade.

Hypotension and bradycardia seen in Case 2 may have been the result of brainstem blockade and hypoxaemia. The ophthalmic theatres in Leicester did not possess pulse oximeters then so we could not assess the degree of hypoxaemia present.

We are disappointed to find that the importance of needle length and positioning of the eye during retrobulbar block is still not widely known, despite another case of apnoea in Leicester and the large number of other cases previously described.

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The incidence of pruritus after epidural morphine

We read with interest the letter from Dr Thangathurai *et al.* (*Anaesthesia* 1988; **43:** 1055–6) wherein the incidence of pruritus after epidural administration of low dose morphine in women for Caesarean sections and men for major abdominal cancer surgery (high dose) were compared.

We have reviewed 52 reports in the literature of the use of epidural and intrathecal opiates, to assess the incidence of pruritus, and it is clear from these reports that opiate-induced itch is more common in pregnant women than in the nonparturient.[1] In patients given morphine (2–5 mg) via the lumbar epidural route, an incidence of 42.7% was reported in pregnant women (593 cases), compared with 8.4% in the nonparturient (3050 cases). Several of the physiological changes of pregnancy could account for the difference. There are high plasma levels of maternal endorphin during labour;[2] there is competition by oestrogen for opiate-binding sites,[3] and the hormone may modify the endogenous opiate content at opiate receptors;[4] there is also more distant spread of morphine (in common with local anaesthetics) within the CSF during labour.[2] The authors did not state whether their cancer patients had received opiates before, but patients with established tolerance to opiates are less likely to develop side effects when epidural opiate therapy is instituted than those who have not.[5] These factors could certainly account for the difference in incidence between the two groups of patients studied by Dr Thangathurai and colleagues, despite the lower dose used in the group with the higher incidence (the pregnant group). An increased incidence of all the side effects of epidural morphine (respiratory depression, nausea and vomiting, pruritus and urinary dysfunction) seems to go hand in hand with the use of higher doses of opiates in patients who have not had opiates, but in those who have developed tolerance the picture is less straightforward, and very high doses may be needed to produce analgesia, and may be administered free of side effects.

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Epidural fentanyl and plasma fentanyl concentrations

The letter from Dr Fairbrass and his colleagues (*Anaesthesia* 1989; **44:** 526) reported that fentanyl cooled to 4°C provided poor epidural analgesia. It is always valuable to publish negative findings to stop others wasting their time.

Their statement (unreferenced) that epidural fentanyl produces plasma levels similar to those seen after intramuscular injection, while the addition of adrenaline produces lower levels, more akin to those after intravenous infusion puzzled me. We have shown[1] that epidural fentanyl in labour produces plasma concentrations that are initially much *higher* than those after the same dose given intramuscularly, while if this dose is given intravenously over 30 minutes, plasma concentrations *exceed* those after epidural fentanyl.[2] What is the source of this information?

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Overdose of ritodrine

A 26-year-old woman in good health who weighed 56 kg underwent routine dilatation and curettage of the uterus, but continued to bleed afterwards from a cervical graze caused by the vulsellum retractor. Her return to theatre for suturing was delayed because she was fed, and in the interim it was decided to prescribe metoclopramide and ranitidine. However, instead of ranitidine 50 mg the patient received a rapid intravenous injection of ritodrine 50 mg. She immediately became flushed and tremulous, her heart rate increased from 90 to 150 beats/minute, and her arterial blood pressure, which had been 120/80 mmHg, became transiently unrecordable before returning in the course of immediate fluid resuscitation to a level of 90/20 mmHg.

It was 3 hours before her diastolic blood pressure exceeded 40 mmHg, but by 6 hours her blood pressure was recorded as 100/60 mmHg. The patient's heart rate returned to its normal level over the same period, having remained at around 130 beats/minuts for the first 3 hours after injection. Baseline serum electrolyte and glucose estimations were not available, but serial measurements of potassium at 1, 2, 3 and 4 hours showed that it did not increase above 2.4 mmol/litre. Blood glucose increased from 10.5 mmol/litre at 2 hours to 14.2 mmol/litre at 4 hours: this decreased without treatment to 3.8 mmol/litre by 12 hours. The hypokalaemia was treated, on the advice of the Leeds Poisons Information Bureau, with 40 mmol KCl infused over 8 hours, and serum potassium was reported at 12 hours as 3.9 mmol/litre. Either coincidentally, or possibly because of the period of hypotension, the cervical wound stopped bleeding and a return to theatre was not required. The patient made a complete recovery.

Ritodrine is a β-adrenoceptor agonist which is used to inhibit uterine contraction in premature labour. It is described in the data sheet as β_2-mimetic in action with some β_1-mediated chronotropic and peripheral vasodilatory effects at therapeutic doses. Its elimination half-life is reported as 1.3–2.0 hours. The dose is usually titrated against response but lies in the region of 150–350 μg/minute. This patient received, therefore, a dose some 150 times greater than recommended. Her cardiovascular and metabolic responses were predictable, and relatively short-lived. The persistent hypokalaemia was treated, but in view of the patient's age and general good health it was not considered necessary to use β-adrenoceptor blockers to treat her tachycardia.

The use of generic drug names did not protect this patient from drug substitution, but more importantly the case re-emphasises that administration of the wrong drug is a recurrent hazard of intravenous therapy which can be minimised only by scrupulous cross-checking of ampoules. The one positive aspect of these incidents is that they do represent one of the few ways in which the effects in humans of massive overdose can be demonstrated, and their management often provides an interesting challenge in applied pharmacology and physiology.

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Fentanyl overdose in a neonate: use of naloxone infusion

This is a report of the management of a large overdose of fentanyl in a neonate, using a continuous infusion of naloxone.

A 3.9-kg, full-term male neonate aged 25 days, was admitted to our hospital with a one-week history of increasing vomiting after each feed. A diagnosis of pyloric stenosis was made and, after correction of metabolic alkalosis, the infant was scheduled for surgery. Anaesthesia was induced, after pre-oxygenation, with thiopentone 5 mg/kg followed by atracurium 0.5 mg/kg. It was intended to give 2 μg/kg of fentanyl at this time; however, despite the fact that the fentanyl syringe was correctly labelled, 100 μg or 26 μg/kg was given in error.

Complete antagonism of neuromuscular blockade was confirmed using a peripheral nerve stimulator at the conclusion of surgery. However, there was little reflex activity, and the infant made no respiratory effort. Accordingly, a dose of naloxone 40 μg was given which failed to produce a response. Further doses of naloxone were then given up to a total of 200 μg, at which point spontaneous breathing started. The trachea was extubated and, after a further bolus dose of 200 μg, an intravenous infusion of naloxone 200 μg/hour was commenced. The infusion was continued for 24 hours, and stopped at 1200 hours the next day. The remaining convalescence was uneventful.

The termination of respiratory depression after small doses of fentanyl is associated with the rapid redistribution of the drug. However, after large doses the duration of respiratory depression is dependent on the elimination half-life of the drug, which is approximately 3 hours in adults and children[1,2] and 5 hours in neonates.[3] Antagonism of narcotic-induced respiratory depression by naloxone is in

contrast invariably brief, and reflects both its rapid redistribution from the brain and its short elimination half-life (64 minutes).[4] Thus, a continuous infusion of naloxone is indicated for the treatment of severe fentanyl overdose.

We followed the recommendations of Tenenbein, who managed two cases of narcotic overdose in infants using naloxone infusions.[5] Our choice of 24 hours for the infusion time was based on the work of Koehntop and colleagues.[3] These authors found that four neonates given 25 or 50 μg/kg of fentanyl during major surgery required ventilatory support for an average of 24 hours after operation. We were also influenced by the fact that the infusion would be terminated at a time when medical staff would be readily available to deal with any recurrence of respiratory depression. We consider, in view of the successful outcome, that continuous naloxone infusion is an alternative to mechanical ventilation in the management of severe fentanyl overdose in the neonate.

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Coughing and laryngospasm with the laryngeal mask

We read with interest the recent article of Brodrick, Webster and Nunn (*Anaesthesia* 1989; **44:** 238–41) on the appraisal of the laryngeal mask airway (LMA). Consistent with our experience, two problems predominated with insertion: firstly, respiratory obstruction attributed to the backward folding of the epiglottis;[1] and secondly, coughing and laryngospasm attributed to inadequate depth of anaesthesia.[2] The former problem was resolved by the use of an introducer; this letter is about the latter problem.

Brodrick and colleagues reported that laryngospasm and (or) coughing complicated insertion in 10% of patients and that similar problems were encountered during recovery. We presume that this arises from stimulation of the laryngeal inlet at light levels of anaesthesia. The supraglottic area of the larynx is simply and safely anaesthetised with superior laryngeal nerve (SLN) block.[3] We have performed this block under light anaesthesia since before the introduction of the laryngeal mask and have encountered no episode of laryngospasm or troublesome coughing. It is useful in the provision of a patent airway in patients who otherwise require light levels of general anaesthesia in combination with regional blockade. Other advantages may include reduced trauma and cardiovascular response during insertion, and fewer problems during recovery and removal of the laryngeal mask. It may also be useful for induction of anaesthesia in patients in whom airway difficulties are anticipated and therefore general anaesthesia contraindicated before the airway is secured.[2]

Superior laryngeal nerve block appears to be a simple and safe procedure with little or short-lived impairment of motor function of the laryngeal inlet. We are currently planning a formal study.

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The use of the fibreoptic laryngoscope to confirm the position of the laryngeal mask

The article by Brodrick, Webster and Nunn (*Anaesthesia* 1989; **44:** 238–41) describes a 10% incidence of respiratory obstruction, which was deemed to be due to downfolding of the epiglottis. We have studied 50 patients, using a fibreoptic laryngoscope to ascertain the final position of the laryngeal mask after insertion and cuff inflation.

The mask was inserted under propofol anaesthesia using a size that was judged appropriate for that patient. No problems were associated with insertion and all patients in this series were breathing spontaneously. The incidence of complete obstruction was 1%, and partial obstruction 10%, which is similar to the study by Brodrick *et al.* However, fibreoptic laryngoscopy revealed that obstruction was not solely due to downfolding of the epiglottis. One complete obstruction was as a result of downfolding of the epiglottis. The second was caused by forward displacement of the posterior cricoid area with infolding of large aryepiglottic folds. The airway was improved after reinsertion of the laryngeal mask (size 4).

The epiglottis was displaced posteriorly in 60% of the patients that were partially obstructed. Large aryepiglottic folds encroached on the laryngeal inlet in the remaining 40%. It appears that the tip of the mask enters the pharyngeal inlet to a greater extent in these patients and, with cuff inflation the aryepiglottic folds are displaced inward and cause partial or complete obstruction. Five of the patients were grossly obese but no airway problems were encountered; two patients were known to be difficult to intubate but the position of the laryngeal mask was satisfactory in these cases.

The oesophagus was clearly visible in three patients. There was no evidence of respiratory obstruction and the mask did not appear to have rotated. The implication of this finding is important in relation to the use of the laryngeal mask in patients at risk from regurgitation of gastric contents and its use for intermittent positive pressure ventilation of the lungs. It reinforces the recommendation by Dr Brain that the laryngeal mask does not protect against aspiration of gastric contents.

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The costs of propofol in day surgery

The potential risks of undergoing anaesthesia in the modern day are brought home by the Committee on Safety of Medicine's recent warning about the occurrence of convulsions and anaphylaxis resulting from the use of propofol.[1] The risks are small (just over one reported adverse reaction for every 10 000 patients anaesthetised) but this potential cost to the patient needs to be borne in mind when anaesthetic agents are selected.

The other major cost of propofol is its high price when compared with the drugs it has largely superseded. An investigation is reported here which determined the extra cost of using propofol in day surgery work in a large district general hospital in South East Thames Health Region in 1988.

A before-and-after design was used to compare the cost of the total anaesthetic drugs' bill for patients for routine day surgery procedures with and without propofol. All prices were adjusted to take account of inflation. This design was chosen because a contemporary comparison group which did not include propofol could not be found.

Table 1. Costs of anaesthetics with and without propofol.

Procedure	Average cost without propofol	Average cost with propofol	Factor: increased cost with propofol
Suction termination of pregnancy	£1.19 $n = 13$	£2.62 $n = 23$	×2.21
Cystoscopy	£1.09 $n = 16$	£3.95 $n = 15$	×2.56
Arthroscopy	£0.94 $n = 6$	£3.10 $n = 9$	×2.97

Study years selected were 1985 and 1987. The total anaesthetic drugs bill per patient was used to allow for factors such as cost differences as a result of different amounts of postoperative antiemetics required in the two groups. Inhalation agents could not be costed because of inadequate recording. The three procedures selected were, suction termination of pregnancy, cystoscopy and arthroscopy. They were chosen because they are standard common procedures. The results are summarised in the Table.

It can be seen that anaesthetics using propofol are about 2.5 times (range 2.21 to 2.97) more expensive than the drugs they superseded. Almost the whole of this price difference is due to the substitution of propofol; differences between the two groups in the cost of other drugs used were negligible. The findings have led to concern among anaesthetists who strive to improve services in an increasingly cost conscious NHS. More money spent on propofol inevitably means less spent on other services in a cost-limited service. It remains to be seen whether the additional costs of propofol will be considered to be outweighed by the benefits to patients. It may be that the recent warnings of the serious adverse reactions to propofol will be enough to tip the balance.

This project was carried out as part of an MSc degree course in the Department of Community Health at the London School of Hygiene and Tropical Medicine.

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A potential complication associated with a tracheal tube with Murphy eye

A side vent in a tracheal tube was designed by Murphy[1] to avoid complete respiratory obstruction if one or more of the openings in the tube were to become occluded by mucus. Gillespie[2] proposed, in addition, another advantage of the Murphy eye which also avoids obstruction from occlusion of the bevelled end resting against the tracheal wall.

Recently, both MacGillivray and Odell,[3] and Nichols and Zornow[4] reported on a potential complication with a fibreoptic bronchoscope which emerged from a tube through the Murphy eye. We encountered a related complication during selective bronchial suctioning using a curved tipped catheter[5,6] several years ago. A suction catheter could neither be advanced nor withdrawn after insertion of the catheter to the tip of the tracheal tube. We removed the tube with suction catheter still in place; as can be seen, the catheter had exited through the eye (Fig. 1). The trachea was then reintubated using a conventional tracheal tube. This complication should not occur if a straight-tipped catheter were used. However, selective bronchial suction is not possible with that kind of suction catheter[5,6] so since then, we have abandoned routine use of tracheal tubes with a Murphy eye.

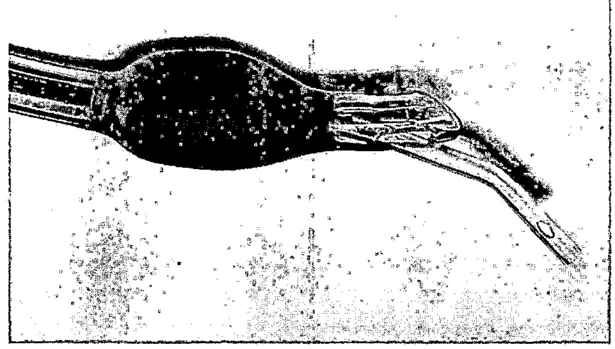

Fig. 1. Curved-tipped suction catheter with a guide mark wedged in the eye of a Murphy tracheal tube.

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Morbidity associated with antigravity suits

Antigravity suits are commonly used in neurosurgical centres where the sitting position is favoured. Fifty-two percent of centres questioned in a survey in 1981 used this position for infratentorial surgery and 31% for cervical laminectomy.[1]

The sitting position is said to give better surgical access to the posterior fossa, and provides improved venous drainage of the skull. The hazards of this position include hypotension, venous pooling in the legs and face and venous air embolism. The antigravity suit has been shown to minimise the haemodynamic changes associated with the sitting position and consequently to decrease the risk of air embolisation.[2]

A 28-year-old obese female (height 1.60 m, weight 69 kg) was anaesthetised for removal of a left-sided acoustic neuroma. The most suitable surgical access to the tumour was deemed to be through a posterior occipital craniotomy and on this occasion the sitting position was used. The antigravity suit (medium size) was inflated to 8 kPa once the patient was positioned in theatre.

She was monitored throughout with ECG, capnograph, pulse oximeter, direct blood pressure measurement and central venous pressure (CVP) via a long line inserted through the basilic vein. Polygeline (500 ml) was transfused, after inflation of the antigravity suit, which increased the CVP to +10 cmH_2O. Her mean arterial pressure remained between 80 and 100 mmHg throughout the procedure.

The antigravity suit was deflated at the end of the operation, the total inflation time was 8 hours, and it was removed in the recovery suite. Large vesicles were seen to have formed in the skin at points which corresponded to folds in the material of the antigravity suit. The vesicles on the medial sides of the thigh had burst and the patient complained of pain over these areas. The lesions resolved over the next 24 hours and left no scars. There was no history obtained of sensitivity to any materials when the patient was subsequently questioned.

This complication of antigravity suits is not already described. It seems likely that the vesicles formed as a direct result of pressure by the suit. Consequently, we now routinely use soft padding (gamgee) beneath the antigravity suit in an attempt to distribute the pressure more evenly.

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Pulse oximetry in mountain rescue and helicopter evacuation

We recently had the opportunity to examine the practicalities of using pulse oximetry to monitor oxygen saturation in a hostile environment: a *simulated* mountain rescue exercise with evacuation by helicopter. The exercise involved two patients, one of which was an unconscious head injured patient rescued from steep terrain by stretcher with a significant journey over rough ground, followed by transport in an RAF SAR Sea King helicopter.

Four pulse oximeters were assessed. Two oximeters were true portable, battery-powered devices, the Engström EOS (Gambro: 10 hours rechargeable battery) and the Catalyst Research MiniOx IV (MIE: 200 hours battery). Two were standard design, mains powered with battery back-up, the Biox 3700 (Ohmeda: 1.5 hours battery) and the Physio Control LifeStat 1600 (Physio Control: 2 hours battery). All were used with finger probes. The assessor (N.P.P.) was present throughout the exercise.

The rescue teams understood the purpose of the devices, after minimal briefing, and were able to use them intuitively, including (without prompting) the use of the pulse indicator to measure systolic pressure. The portable devices were of a practical size to be used on the initial rescue and carry.

The teams found the oximeters valuable in the assessment of adequacy of the airway and breathing during manoeuvres over difficult terrain: assessment stops every few feet were not required. The continuous read-out and audible tone freed a team member, and allowed him to attend to other aspects of treatment.

The LED display, used in the Physio Control oximeter, was almost invisible in strong sunlight; the other oximeters utilised LCD displays, which were readable in all situations. A night exercise might alter this comment. All four oximeters functioned normally in the Sea King helicopter, despite extreme vibration. This observation was unexpected. The pulse waveform displayed by the Biox 3700 showed no spurious pulsation. The pulse indicators allowed measurement of systolic blood pressure in the helicopter. Noise and vibration in this environment, makes the measurement of blood pressure using palpation or auscultation impossible.

The Miniox IV was the preferred model tested. It is small in size, rugged in construction (particularly at the lead-instrument interface), has a long battery life of 200 hours on a PP3 alkaline battery, and an LCD display. The mountain rescue teams concluded that pulse oximetry would be of significant help in monitoring in adverse conditions. In this context, the MiniOx IV costs little more than a two-way VHF radio. Further trials, particularly in wet, cold and dark conditions would be required.

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An erroneous pulse oximeter reading

A 30-year-old ASA 1 patient was to undergo posterior fossa craniotomy for excision of a pineal tumour.

He was anaesthetised with a standard neuroanaesthetic method using relaxants and hyperventilation of the lungs. Vascular access included the siting of a right radial artery cannula for direct arterial pressure monitoring; the insertion of this caused the loss of several millilitres of blood.

The patient was turned into the prone position and transferred to theatre. A finger probe for a Datascope Accusat pulse oximeter was placed on the right index finger in theatre. The pulse rate according to the pulse oximeter corresponded to the heart rate from the ECG, but the $Sa{O_2}$ reading was 90%. However, there was no evidence of cyanosis, both lungs were well ventilated and an oxygen analyser showed the inspired oxygen to be 35%.

The oximeter finger probe was removed and the index finger was found to be covered in dried blood. The finger was cleaned with spirit and dried with gauze. The $Sa{O_2}$ reading was 99% when the probe was reapplied.

Many pigments, such as nail varnish do not significantly affect pulse oximeter readings, but, as would be expected external haemoglobin does, and therefore dried blood should be removed from pulse oximeter probe sites.

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The high-risk register

We write to describe a computer program which alerts staff to patients known to be at high risk from anaesthesia.

Limited computerised records have been kept in this department since 1982. They are based on the Anaesthetic Records System programme designed by one of the authors (J.D.A.) in association with the Eaton Partnership. This was originally used for workload analysis, but the data include some clinical information such as ASA gradings and problems encountered as a result of anaesthesia. It was decided late in 1986 to utilise the latter information and extend and adapt it in order to produce a register of patients known to have encountered problems during anaesthesia or to have abnormalities which might lead to problems. Thus was created the department's register for patients at high risk from anaesthesia.

The register lists the patients' names and anaesthetic problems in central index number order. It can also list in alphabetical order. It can be accessed by computer in the principal anaesthetic areas, with a printed copy in every anaesthetic location. Every case scheduled for anaesthesia is checked against the register. This can be done by any member of the theatre staff; for elective cases it is done by the theatre secretary when typing the lists.

The register is confined to patients with conditions which present a particular problem for anaesthesia. Most general medical conditions are not included; these would normally be discovered on history-taking, examination or from the patient's medication; furthermore, the severity and treatment of such disorders varies with time so the degree of extra risk may change. The main groups included are: previous difficult intubation, known hypersensitivity to an anaesthetic drug, history of jaundice after anaesthesia, malignant hyperpyrexia susceptibility, plasma cholinesterase abnormality, haemoglobinopathies, porphyria and cardiac arrest during a previous anaesthetic.

Most patients are added automatically from the department's computer records. This is supplemented by information on patients (and on tested relatives) from the pathology laboratories. Patients may be added to the register by any anaesthetist, but removed only by consultants. It is departmental policy that no patient on the register (or with a condition which will lead to placement on it) may be anaesthetised without prior discussion with a consultant.

The register is not a substitute for the anaesthetist's pre-anaesthetic assessment. It is supplementary to this and acts as an early-warning system of certain problems, some of which might easily be missed during the assessment, especially if the notes are not available or past anaesthetic records are missing.

The register will always be incomplete because patients can only normally be added if they have had an anaesthetic in this district since 1982. A few patients are removed, for example if intubation is no longer difficult because of dental clearance, but the register is growing steadily.

It cannot be determined for certain whether the register has actually reduced anaesthetic mortality or morbidity, but it has resulted in alterations to anaesthetic technique or avoidance of general anaesthesia in individual cases. It is hoped that this has increased the safety margin for the patient.

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Obstetric anaesthetic records

Our colleagues in departments of Pathology and Medical Records depend on the selfadhesive Patient Identification label to reduce the amount of handwriting. My use of printed, sticky labels fixed to the obstetric anaesthetic record chart has convinced me that they confer many benefits.

The general anaesthetic for Caesarean section has many features which make it suitable for this approach to record keeping. The patients are relatively similar, the induction procedure is standardised and the monitoring does not vary since usually a single theatre is used. When vigilance is the first priority, the single-handed anaesthetist tends to perform a high speed scribble (so appreciated by plaintiffs' counsels in recent years!) when making his record. Rapid scribbling reduces distraction time, but only marginally so, and at the expense of reduced legibility and increased medicolegal risk.

Three printed labels are used. The first is for the induction, intubation and ventilation details, the second for monitoring equipment and the third for postoperative instructions and the name of the anaesthetist. Details of drug and fluid administration and recording of monitored

values are added to complete the intra-operative record, otherwise I am free to resume observation of the patient.

The labels are composed using the 'Address Label' function in the mailing-list facility available in most word processors. The actual labels are standard 70 × 36 mm address labels and can take up to eight lines of text.

If the address label is adapted in this way, each obstetric anaesthetist could have his own customised set of labels which can be updated at will. The anaesthetist can simply revert to handwriting if an unusual technique is employed.

The selfadhesive label concept is cheap, flexible and practical. Most departments already have both the necessary hardware and software. The sticky label passes the ultimate test of any computer application. It does the job better and more quickly than pen and ink so it frees its user to concentrate on the main task with vigilance.

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Mediastinal trauma—or not?

The diagnosis and successful management of road traffic accident victims with injuries to the major thoracic vessels requires a high level of suspicion, careful clinical examination and specialist radiological facilities. We report a case where perhaps too high a level of suspicion, along with a misdiagnosed clinical sign, led to an expensive, invasive and unnecessary radiological investigation.

A 32-year-old male was admitted to the intensive care unit after a road traffic accident in which he was hit by a motor-cycle. He had a severe head injury (Glasgow coma scale 3) and his lungs were hyperventilated electively after wound debridement and suturing. Thirty-six hours after admission he was noted to have a bruise over the upper sternum, possibly a widened mediastinum on chest X ray, and a decrease in haemoglobin concentration from 13 g/d litre to 9 g/d litre. He was otherwise stable with persistent primary brain injury. The neurosurgeons discussed the case with the thoracic surgeons and decided that an arch aortogram was indicated. This was normal.

A second neurosurgical patient whose lungs were ventilated after a subarachnoid haemorrhage was noted to have an identical bruise over the upper sternum despite the absence of a history of major trauma. We then realised that these bruises were secondary to the technique used by one of the neurosurgical registrars of testing for a reaction to pain by pressing his knuckles hard against the patient's sternum. We now find out if this technique has been used on a patient before diagnosing chest/mediastinal trauma!

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When in doubt, take it out ... the epidural catheter

We have recently cared for a previously healthy 21-year-old primipara who suffered complications as a result of an epidural sited for pain relief in labour.

The first insertion of the epidural catheter appeared to be uncomplicated. Preloading with 1 litre of compound sodium lactate solution, followed by a test dose of 3 ml 0.5% plain bupivacaine were given uneventfully. Four millilitres of 0.5% plain bupivacaine given 8 minutes later resulted in significant hypotension which eventually responded to a 2-litre hydroxyethyl starch infusion (HES) and incremental ephedrine to a total dose of 19 mg. Four further top-ups, each of 5 ml 0.25% plain bupivacaine were given in the next 12 hours. Hypotension, that required further crystalloid infusion, followed each top-up. There was no other evidence of subarachnoid or intravenous injection of local anaesthetic, other than hypotension.

Labour failed to progress, and the quality of analgesia was inadequate to allow for Caesarean section. General anaesthesia was necessary. The patient had received 11 litres of intravenous fluid during the 12 hours prior to the general anaesthetic; 9 litres was crystalloid, and there was a positive balance of 7.5 litres.

She was moved to the recovery area after uneventful anaesthesia, and delivery of a healthy infant (Apgar 9), where she became cyanosed and dyspnoeic. She required transfer to the Intensive Therapy Unit. A clinical diagnosis of pulmonary oedema was confirmed by chest X ray. Blood gases breathing air showed Pa_{O_2} 6.4 kPa with a Pa_{CO_2} of 4.4 kPa. Her serum albumin was 23 g/litre and the colloid oncotic pressure 18 mmHg (normal 21–26 mmHg). The patient was treated with oxygen, intravenous albumin and diuretics. Her respiratory problems rapidly resolved.

It is clear that the epidural was responsible for the hypotension. The location of the epidural catheter tip cannot be known for certain, but the hypotension after relatively small top-ups suggest either subdural or subarachnoid placement or migration. Treatment of hypotension followed standard practice. Unfortunately the positive cumulative fluid balance passed unnoticed, and its significance went unrecognised. Pulmonary oedema followed the resumption of sympathetic tone once the block had become ineffective.

Three standard anaesthetic textbooks[1-3] and two authoritative texts of obstetric anaesthesia[4,5] provide a wealth of information on the diagnosis and management of complications associated with epidural analgesia, in particular, inadvertent subarachnoid or intravenous injection and catheter tip migration. There are a number of lessons to be learnt from this patient, but these texts do not comment upon the implications and importance of progressive fluid loading, nor, more importantly, do they give one obvious piece of advice taught to all trainees learning tracheal intubation: 'when in doubt, take it out'.

We suggest that this aphorism should be as vigorously applied to the practical management of epidural analgesia, or, indeed, to any other invasive procedures.

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Tracheostomy in obstetric practice—how about the laryngeal mask airway?

While heartily agreeing with your correspondents in deploring the suggestion that a woman who is extremely difficult to intubate should be given an elective tracheostomy during pregnancy, and concurring entirely with their recommendations that such a woman should be offered a regional block should surgical intervention prove necessary,[1,2] I am nonetheless amazed that no-one has mentioned the alternative of using the laryngeal mask-airway.[3,4] Regional techniques are preferable to general anaesthesia in the majority of parturients, and with experience the need for general anaesthesia becomes extremely rare, but use of the laryngeal mask airway represents the ideal fall-back position, in the unlikely event that general anaesthesia or resuscitation should be needed following regional blockade.

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The collator of this section is Dr L. Kaufman, MD, FFARCS, 145 Harley Street, London W1N 2DE. Dr Kaufman is prepared to provide on request, and at a modest charge, a new additional service to our readers. References from January 1984 have been entered on data base. The data are held on Dbase II, cpm 86 and on Dbase III, MsDos and are available on disc, together with a program providing search facilities. Enquiries direct to Dr Kaufman at the above address please.

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Treatment and medication

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Anaesthesia, 1989, Volume 44, pages 876–877

Obituaries

Brown, Ian Michael, MB, BChir, MRCS, LRCP, FFARCS, DA, formerly Consultant Anaesthetist to Stockport and Buxton Hospital. Qualified from University of Cambridge in 1953.
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Sampaio, Margarida, M. M. V., MB, ChB, FFARCSI, DRCOG, formerly Senior Registrar at Royal Victoria Hospital in Belfast. Qualified from University of Glasgow in 1980.

International congress calendar

1989

4-6 October. Rome, Italy. *International Symposium on Survival under Critical Life Conditions.*
Information: Organizing Secretariat, Fondazione Giovanni Lorenzini, Via Monte Napoleone 23, 20121 Milan, Italy.

4-7 October. Tunis. *Third Pan Arab Congress on Anaesthesia and Intensive Care.*
Information: Secretary, P.O. Box 15404, Marka, Amman, Jordan.

14–18 October. New Orleans. *American Society of Anesthesiologists Annual Meeting.*
Information: Executive Secretary, 515 Busse Highway, Park Ridge, Illinois 60068, USA.

18–20 October. Barcelona. *11th Congress of Chronical Roncopathy.*
Information: BRP, Edificio Layetana C-Pau Claris, 138, 08009, Barcelona, Spain.

19–21 October. Padova, Italy. *1st International meeting on Pediatric Care.*
Information: Sistema Congressi, Via Jappelli 12, 35121 Padova, Italy.

21 October. London. *Postgraduate Study Day, Association of Anaesthetists of Great Britain and Ireland and the College of Anaesthetists.*
Information: College of Anaesthetists, 35 Lincoln's Inn Fields, London, WC2.

26–28 October. Berlin. *3rd International Steglitz Symposium on Clinical Aspects of O_2 Transport and Tissue Oxygenation.*
Information: M. Specht, Klinik für Anaesthesiologie und Operative Intensivmedizin, Klinikum Steglitz FU-Berlin, Hindenburgdamm 30, D-1000 Berlin 45.

18–22 October. Tokyo and Kyoto. *6th World Congress for Bronchology.*
Information: Dr M. Niitsuma, Secretary General, 6th World Congress for Bronchology, Department of Surgery, Tokyo Medical College, 6-7-1 Mishishinjuku, Shinjuku-ku, Tokyo 160 Japan.

3–5 November. Toronto, Canada. *Paediatric Anaesthesia Conference.*
Information: Paediatric Anaesthesia Conference, The Hospital for Sick Children, 555 University Avenue, Toronto, Ont. M5G 1X8, Canada.

5–10 November. Sao Paulo. *36th Brazilian Congress of Anesthesiology.*
Information: Congress Secretariat, Dr R.S. Mathias, Rua Caiubi 666, Sao Paulo, Brazil 05010.

11–17 November. River Rhine. *International Symposium to commemorate '60 years of surfactant research'.*
Information: Professor B. Lachmann, Department of Anesthesiology, Erasmus University, POB 1738, 3000 DR Rotterdam, The Netherlands.

28 November–1 December. Manila. *6th ASEAN Congress of Anaesthesiologists.*
Information: P.O. Box 4486, Manila, Philippines.

9–13 December. New York. *Forty-third Postgraduate Assembly in Anesthesiology.*
Information: Kurt G. Becker, Executive Director, The New York State Society of Anesthesiologists Inc., 41 East 42nd Street, Suite 1605, New York 10017.

16–23 December. Vail, Colorado. *Current issues in Medicine.*
Information: Professional Seminars Inc., P.O. Box 012318, Miami, FL 33101.

1990

13–20 January. Virgin Islands. *8th Annual Symposium on Clinical Update in Anesthesiology.*
Information: Dr G. Silvay, Dept. of Anesthesiology, Box 1010, Mount Sinai School of Medicine, 1 Gustave L. Levy Place, New York, NY 10029-6574.

3–10 February. Colorado. *Sixteenth Annual Vail Conference in Anesthesiology.*
Information: Sonja Craythorne, Professional Seminars, P.O. Box 012318, Miami, Florida 33101.

10–17 February. Colorado. *Fifteenth Annual Vail Symposium in Intensive Care.*
Information: Sonja Craythorne, Professional Seminars, P.O. Box 012318, Miami, Florida.

24–26 February. New Orleans. *Mardi Gras Anaesthetic Course.*
Information: Professor A. Grogono, Tulane University Medical Centre, 1430 Tulane Avenue, New Orleans, Louisiana 70112, USA.

1–3 March. Mainz, Germany. *Second Postgraduate Course on Anaesthesia, Intensive Care Medicine, Emergency Medicine and Pain Treatment.*
Information: Prof. Dr W. Dick, Klinik für Anaesthesiologie, Langenbeckstr 1, D 6500 Mainz.

10–14 March. Honolulu. *64th Congress of the International Anaesthesia Research Society.*
Information: Professor E.A. Moffitt, IARS, 3645 Warrensville Center Road, Cleveland, Ohio 44122, USA.

17–23 March. Durban, South Africa. *National Anaesthetic Congress.*
Information: Professor D.A. Rocke, 1990 Anaesthetic Congress, Department of Anaesthetics, P.O. Box 17039, Congella 4013, South Africa.

27–30 March. Brussels, *10th International Symposium on Intensive Care and Emergency Medicine.*
Information: Dr J.L. Vincent, Dept. of Intensive Care, Erasme University Hospital, Route de Lennik 808, 1070 Brussels, Belgium.

4–6 April. Glasgow. *Junior Anaesthetists' Group of the Association of Anaesthetists of Great Britain and Ireland. Linkman Conference and Annual Scientific Meeting.*
Information: Honorary Secretary, Association of Anaesthetists of Great Britain and Ireland, 9 Bedford Square, London, WC1B 3RA.

13–19 May. Wellington, New Zealand. *Faculty of Anaesthetics and the Royal Australasian College of Surgeons Annual General Meeting.*
Information: Administrative Officer, Faculty of Anaesthetists, RACS, Spring Street, Melbourne, Victoria 3000.

5–8 June. Amsterdam. *5th European Congress on Intensive Care Medicine 1990.*
Information: Secretariat, c/o Holland Organizing Centre, Lange Voorhout 16, 2514 EE, The Hague, The Netherlands.

15–19 June. Vancouver. *47th Annual Meeting of the Canadian Anaesthetists' Society.*
Information: Ms Ann Andrews, CAS 187, Gerrard St. E, Toronto, Ontario M5A 2E5 Canada.

5–8 September. Budapest, Hungary. *3rd International Symposium on Anesthesia for Cardiac Patients.*
Information: Dr G. Silvay, Department of Anesthesiology, Box 1010, Mount Sinai School of Medicine, 1 Gustave L. Levy Place, New York, NY 10029-6574, USA.

9–15 September. Warsaw. *VIIIth European Congress of Anaesthesiology.*
Information: The Organising Committee, VIIIth European Congress of Anaesthesiology, c/o The Polish Society of Anaesthesiology and Intensive Therapy, ul. Kasprzaka 17a, 01-211 Warsaw, Poland.

23–28 September. Seoul. *8th Asian/Australasian Congress of Anaesthesia.*
Information: Department of Anesthesiology, Seoul National University Hospital, 28 Yungun-Doug, Chongro-Ku, Seoul 110.

26–28 September. Manchester. *Linkman Conference and Annual Scientific Meeting.*
Information: The Honorary Secretary, Association of Anaesthetists of Great Britain and Ireland, 9 Bedford Square, London, WC1B 3RA.

19–23 October. Las Vegas. *American Society of Anesthesiologists Annual Meeting.*
Information: Executive Secretary, ASA, 515 Busse Highway, Park Ridge, IL 60068, USA.

5–10 November. Sao Paulo. *36th Brazilian Congress of Anesthesiology.*
Information: Dr R. Mathias, Rua Caiubi 666, Sao Paulo, Brazil 05010.

18–21 November. Kyoto, Japan. *4th International Symposium on the Pain Clinic.*
Information: 4th WSPC Secretariat, Department of Anesthesiology, Osaka Medical College, c/o Inter Group Corp., Shohaku Bldg., 6–23 Chayamachi Kita-ku, Osaka 530, Japan.

5–9 December. San Juan. *15th Caribbean Symposium in Anaesthesia and Related Fields.*
Information: Miguel Colon-Morales, GPO Box 4547, San Juan, Puerto Rico 00936.

8–12 December. New York. *Forty-fourth Postgraduate Assembly in Anesthesiology.*
Information: Kurt G. Becker, Executive Director, The New York State Society of Anesthesiologists Inc., 41 East 42nd Street, Suite 1605, New York 10017.

1991

8–12 March. San Antonio. *65th Congress of the International Anesthesia Research Society.*
Information: Emerson A. Moffitt, IARS, 3645 Warrensville Center Road, Cleveland, Ohio 44122, USA.

3–5 April. Oxford. *Junior Anaesthetist's Group of the Association of Anaesthetists of Great Britain and Ireland Linkman Conference and Annual Scientific Meeting.*
Information: Association of Anaesthetists of Great Britain and Ireland. 9 Bedford Square, London, WC1B 3RA.

9–12 May. Washington DC. *6th International Dental Congress on Modern Pain Control.*
Information: American Dental Society of Anesthesiology, Inc., 211 E. Chicago Avenue, Suite 948, Chicago, IL 60611.

21–25 June. Quebec City. *48th Annual Meeting of Canadian Anaesthetists' Society.*
Information: Ms Ann Andrews, CAS, 187 Gerrard St. E., Toronto, Ontario M5A 2E5, Canada.

11–13 September. Harrogate. *Linkman and Annual Scientific Meeting of Association of Anaesthetists of Great Britain and Ireland.*
Information: Association of Anaesthetists of Great Britain and Ireland, 9 Bedford Square, London, WC1B 3RA.

26–30 October. San Francisco. *American Society of Anesthesiologists Annual Meeting.*
Information: Executive Secretary, ASA, 515 Busse Highway, Park Ridge, IL 60068, USA.

7–11 December. New York. *Forty-fifth Postgraduate Assembly in Anesthesiology.*
Information: Kurt G. Becker, Executive Director, The New York State Society of Anesthesiologists Inc., 41 East 42nd Street, Suite 1605, New York 10017.

1992

29 March–2 April. Atlanta, Georgia. *The Third International Symposium on the History of Anaesthesia.*
Information: R.K. Calverley, Medical Center, University of California, 225 Dickinson Street, San Diego, California CA 92103, 1990, USA.

14–19 June. The Hague. *10th World Congress of Anaesthesiology.*
Information: Dr Harm Lip, Nilantsweg, 99, 8041 AR Zwolle, Netherlands.

17–21 October. New Orleans. *American Society of Anesthesiologists Annual Meeting.*
Information: Executive Secretary, ASA, 515 Busse Highway, Park Ridge, IL 60068, USA.

1993

15–17 September. Glasgow. *Joint Meeting between the Association of Anaesthetists of Great Britain and Ireland and the Canadian Society of Anaesthetists.*
Information: The Honorary Secretary, Association of Anaesthetists of Great Britain and Ireland, 9 Bedford Square, London, WC1B 3RA.

9–13 October. Washington DC. *American Society of Anesthesiologists Annual Meeting.*
Information: Executive Secretary, ASA 515 Busse Highway, Park Ridge, IL 60068, USA.

Anaesthesia, 1989, Volume 44, page 878

Safety Action Bulletin

89 (28) Medical gas cylinders: safety and care in their storage, handling and use

An oxygen medical gas cylinder was refilled with nitrogen and labelled by hand contrary to colour coding on the cylinder itself. This was done on hospital premises. The cylinder was then placed in a hospital medical gas cylinder store but a serious incident was fortunately avoided. See HEI163.

89 (29) Ohmeda XL Anaesthetic Machine: configuration of control

Anaesthetists and other relevant theatre staff should be aware that the relative position of these flow meter controls have been changed. The oxygen and nitrous oxide controls are now adjacent and all other gases are to the right of them.

Carbon dioxide flowmeters on anaesthetic machines (SAB (89) 36)

Serious injury to a patient resulted from the inadvertent administration of carbon dioxide during an anaesthetic. All anaesthetic machines should be checked for correct function before use. This check should verify the position of all flowmeter valves and confirm that they are closed. Cylinders of carbon dioxide should be turned off when they are not in use.

A carbon dioxide cylinder should be fitted to the anaesthetic machine only at specific request of the anaesthetist and removed after use. Users should also ensure that the carbon dioxide yoke is fitted with a nonreturn valve or a gas-tight blanking plug. If either of these precautions are not taken it is possible for loss of anaesthetic gas and vapour to occur through the flowmeter valve if this is left open.

Erratum

Anaesthesia, 1989, Volume 44, pages 611–612

Intra-ocular pressure changes during rapid sequence induction of anaesthesia

Drs Edmonson, Lindsay and Chew replied to a criticism from Dr Mirakhur on this subject and the error contained in their letter was unfortunately published. A sentence in their letter should read:

Suxamethonium given in isolation produces a mean increase in IOP of 7 mmHg: intubation of 5 to 30 mmHg; and coughing an increase of up to 40 mmHg. Our study demonstrated that after administration .... the increase in IOP was a mean of 1 mmHg. This would therefore indicate a relatively low risk of vitreous extrusion.

Contents: Anaesthesia, vol. 44, no. 10, October 1989

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The Association of Anaesthetists of Great Britain and Ireland
Patron: HRH The Princess Margaret, Countess of Snowdon

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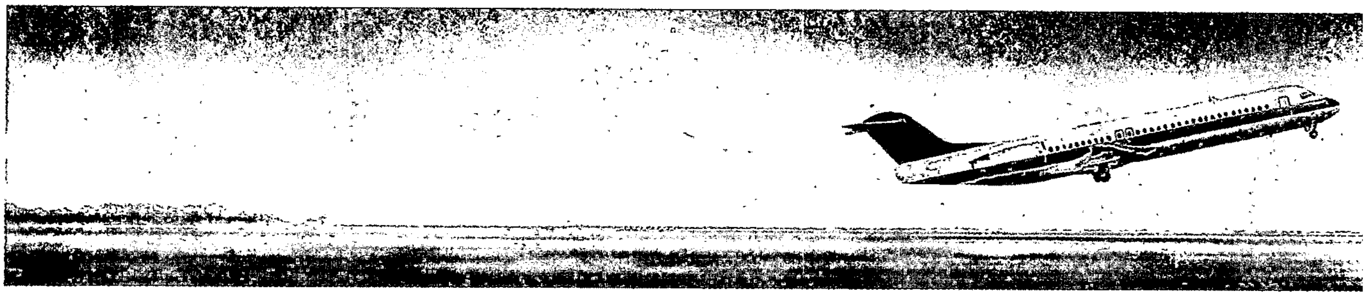
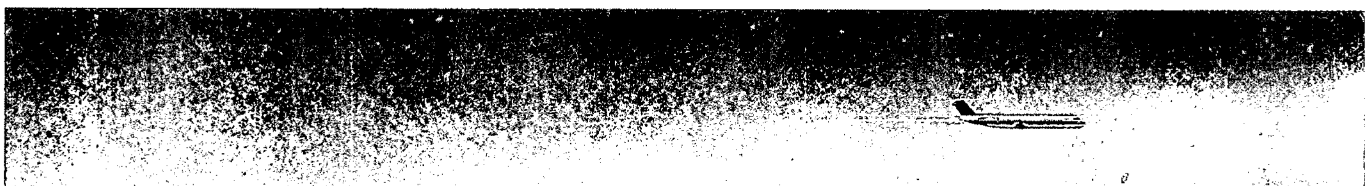
If you're using 'Diprivan' for induction only,



you're not making the most of 'Diprivan'.



The smooth induction and rapid, clear headed recovery obtained with 'Diprivan' are now widely recognised. But there are other benefits too:

* Stable, easily controlled maintenance.
* Few excitatory effects.
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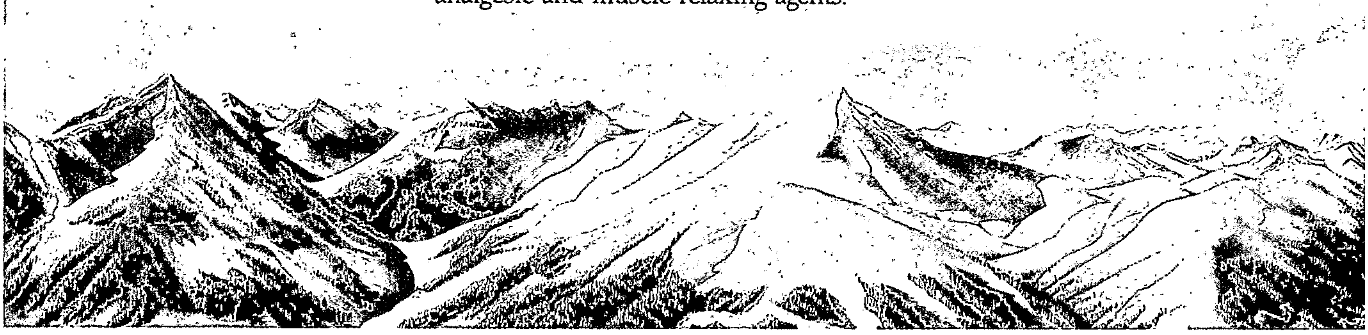

▼ **'DIPRIVAN' PRESCRIBING NOTES Consult data sheet before prescribing. Use** Induction and maintenance of general anaesthesia for surgical procedures lasting up to one hour. **Presentation** Ready to use, isotonic, aqueous emulsion containing 10mg/ml propofol in a vehicle containing soybean oil and purified egg phosphatide. **Dosage and administration Induction** Titrate against response using approximately 4ml every 10 seconds in healthy adults and 2ml every 10 seconds in patients of ASA grades 3 and 4. Patients under 55 years are likely to require 2.0 to 2.5mg/kg, older patients may require approximately 20% less. **Maintenance** Usually 0.1 to 0.2mg/kg/min (6 to 12mg/kg/hr). Continuous infusion may require slightly higher rates for 10 to 20 minutes after induction. Alternatively repeat bolus injections of 25 to 50mg (2.5 to 5.0ml) may be used. **Paediatric Use** No experience in children or in mothers who are breast feeding. **Precautions:** Do not mix prior to administration with other agents or infusion fluids. Hypotension and transient apnoea may occur during induction. Occasionally, hypotension may require use of i.v. fluids and lower rate of administration during maintenance. Apply caution in cardiac, respiratory, renal or hepatic impairment; in hypovolaemic or debilitated patients; and in disorders of fat metabolism or conditions where lipid emulsions should be used cautiously. On general principles, do not use in pregnancy except for termination. **Side effects** Bradycardia responsive to atropine has been reported. Epileptiform movements have occurred rarely in temporal relationship to 'Diprivan' but no causa relationship has been established. Nausea, vomiting and headach in a small proportion of patients. Very rarely, a clinical syndrome which may include bronchospasm and erythema accompanied by hypotension, has been reported with 'Diprivan'. Pain on injection in a proportion of patients. Venous sequelae are rare. Minimal evidence of excitation on induction. **Product licence number** 29/0190 **Basic NHS cost** £3.98 per 20ml ampoule. Lower price available fo hospitals. **'Diprivan'** is a trademark.

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Make the most of it

Anaesthesia, 1989, Volume 44, pages 879–880

Editorial

Infusions of analgesics, sedatives and muscle relaxants in patients who require intensive care

Patients who require artificial ventilation of their lungs during a period of intensive care may need opioids for pain relief, respiratory depression and antitussive effects. Sedative drugs may be needed in addition to allay anxiety, and muscle relaxants are employed in specific circumstances to prevent patients struggling against the ventilator. Originally these were given by intravenous bolus injection, primarily because only 'long acting' drugs such as morphine, diazepam and pancuronium were available and there was little need to administer them using continuous intravenous infusions. In the late 1970s 'short acting' induction agents (Althesin and etomidate) became available for use during anaesthesia. These were investigated as agents to be given by prolonged continuous intravenous infusion to the critically ill, and produced a controllable depth of sedation from which patients would awake rapidly when they were discontinued. Both drugs have now been withdrawn for this purpose, but other drugs with a similar reported duration of action have been investigated for this use. They include chlormethiazole, midazolam, alfentanil, vecuronium, atracurium and propofol.

Infusion of these 'short-acting' drugs has gained widespread acceptance which might indicate improved efficacy compared to bolus administration but little scientific evidence is forthcoming to support this view. There is little doubt that infusion of any of these drugs at a sufficient dose is an effective way to achieve analgesia, sedation or neuromuscular paralysis. However, problems with these drugs arise when the infusions are stopped if the drug effects do not end as predicted by their known pharmacokinetics. Prolonged sedation of several days duration was reported first with midazolam[1] and subsequently chlormethiazole.[2] Similar unexpected prolongation of effect is described in one patient after an infusion of alfentanil[3] and in several patients with renal failure after infusions of vecuronium.[4]

The prolonged duration of effect of the drugs may not be recognised and may mislead clinicians into thinking that cerebral impairment has occurred as a result of the original illness. This may result in unnecessary investigations such as cranial computerised tomography, and inappropriately influence major decisions about treatment. Furthermore, unnecessary excessive sedation may result in difficulties in weaning from artificial ventilation, cardiovascular and renal dysfunction. Opiates may prolong gastrointestinal stasis, and unnecessarily increase the duration of parenteral nutrition. Additionally white cell function was shown to decrease in mice after large doses of morphine[5] and *in vitro* studies have shown an inhibition of human leucocyte phagocytic activity when exposed to morphine.[6]

Why do these drugs behave in such an unexpected manner in some patients? Much of the early work on the pharmacokinetics and pharmacodynamics of drugs is performed on healthy experimental animals, volunteers and ASA class 1 or 2 patients. These groups bear little resemblance to the aged, critically ill patient with multisystem disease found in most intensive care units. It is not surprising that when such patients are studied profound reductions in the ability to eliminate drugs or their metabolites is demonstrated. This reduced ability may be the result of renal disease, age, liver dysfunction or sepsis.[7] Pharmacogenetic abnormalities have been demonstrated which affect 6% of the population who are slow metabolisers of midazolam.[8] There is evidence which suggests a similar abnormality for alfentanil in a similar proportion of patients[9] although this has recently been questioned. These pharmacogenetic abnormalities may be of little importance during and immediately after anaesthesia, since termination of drug effect is usually due to redistribution. However, when the drug is infused for days, a significant prolonged effect may be expected since the drug is no longer redistribution-limited but now elimination-dependent.

What is the answer to the effects of prolonged duration of action? Specific antagonists now exist for opiates (naloxone) and benzodiazepines (flumazenil). These may be given by bolus injection but because of their short duration of action, they may need to be administered by repeated bolus injection[10] or continuous intravenous infusion. Both naloxone and flumazenil are expensive and infusions of both drugs may be costly. Are costly infusions of short-acting reversal agents really the answer to the prolonged effects seen after equally costly infusions of 'short-acting' drugs? Is it not time that analgesics, sedatives and muscle relaxants were used more efficiently? One answer is to monitor and record the effect of the drug and alter the dose accordingly. Monitors of depth of sedation and analgesia include the cerebral function monitor and changes in evoked potentials. These are not in widespread use because they are expensive, lack specificity and are currently difficult to interpret, particularly in the critically ill. We have for the past 5 years used a simple scale[11] which allows us to measure and record the effects of sedation each hour where necessary. Its use has proved invaluable in the improvement of patient care, although the scale can, we consider, still be improved.

Another answer may be to recognise the need for analgesia, sedation or muscular paralysis and give an appropriate drug by bolus injection to produce that effect. The result is assessed and when the effect of the drug is wearing off a repeat bolus dose is given and this procedure repeated several more times. Only if the time interval between the doses is short is an infusion started. Used in this way the need for the drug *and* its method of administration are assessed. This method minimises the risk of overdose and accumulation but has the disadvantage that in some patients unwanted effects may occur, if the drug's effect wears off unexpectedly or before a further dose can be given, such as sudden agitation, patient discomfort, cardiac instability and high airway pressures. If an infusion is necessary then

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once it is started, unless it is obvious the patient is not excessively sedated, it should be stopped or the dose substantially reduced each day and the patient allowed to recover to ensure accumulation does not occur. Should the infusion require to be restarted the need to give the drug at the same rate should be questioned.

The final option is to produce drugs that do not accumulate. Propofol is currently under investigation in this field. Continuous infusion of propofol in the critically ill is now increasingly advocated, because its short half-life and lack of accumulation provide many of the properties of the ideal sedative. Initial experience has shown it to be an effective and easily controlled sedative with rapid recovery on termination of infusion.[12] However, adverse cardiovascular effects are reported both in fit patients[13] and in the critically ill.[14] Briggs *et al.*[15] have shown that the concurrent administration of fentanyl with propofol alters its pharmacokinetics and leads to an increase in its plasma concentrations. Will interactions with fentanyl and perhaps other drugs lead to difficulties with its use during prolonged infusion?

Is any drug without risk? Recent studies suggest that during prolonged infusions accumulation of atracurium and its neurotoxic metabolite laudanosine is unlikely,[16] even in the presence of renal failure. However, clinicians must remain aware of unsuspected adverse effects with new drugs, particularly drug reactions. For example local anaesthetics accumulate in critically ill patients and lower the convulsive threshold. In humans, will this lower the toxic threshold for laudanosine?

If parenterally administered sedatives cause so many difficulties perhaps we should examine the inhaled agents again. Isoflurane was recently studied for sedation of critically ill patients for a 24-hour period and it shows promise as an agent for short-term use.[17] Nitrous oxide was, in the past, widely used for this purpose until bone marrow suppression was recognised. Will isoflurane also be found to have as yet unrecognised difficulties when used for longer periods?

Anaesthetists have always been in the forefront of modern day intensive care bringing their knowledge and skills acquired in the operating theatre to the critically ill. With the increased ability to support patients' lives for prolonged periods, pharmacological knowledge acquired from anaesthetic practice needs to be carefully interpreted when applied to the altered pathophysiology of the critically ill. *In somno securitas* remains an appropriate motto for the Association but, like all good things, too much can be bad!

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Cambridge CB2 2QQ

G.R. Park
P.A. Gray

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Editorial notices

Anaesthesia News

Copy for *Anaesthesia News* should be sent to the Editor as and when it becomes available. It is hoped that publication will occur within 2 months of receipt.

Manuscripts must be submitted in accordance with the internationally recognised *Uniform requirements for manuscripts submitted to biochemical journals* (*British Medical Journal* 1979: **1:** 432 5). Details will be found in the Notice to Contributors to *Anaesthesia* at the end of this issue.

Anaesthesia, 1989, Volume 44, pages 881–884

The detection of intra-operative myocardial ischaemia

Preliminary experience with the right-sided precordial lead

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Summary

The value of monitoring the right precordial lead, V4R, to detect peri-operative ischaemic events during coronary artery surgery was studied in 60 patients. Thirty-four patients had only left-sided coronary disease (Group 1). The other 26 patients had both left-sided occlusive coronary artery disease and significant right-sided occlusive lesions on coronary angiography (Group 2). Lead sensitivity was estimated, assuming that all ST segment changes were true positive responses. Sensitivity using a single lead was greatest for lead V5 in the two groups (73% for Group 1 and 69% for Group 2). Sensitivity in Group 1 for lead II was intermediate (55%), whereas sensitivity for lead V4R was only 9%. In Group 2, on the other hand, lead V4R was 54% sensitive and lead II only 31%. The combination of leads V4R and V5 increased the sensitivity to 92% in Group 2, whereas lead II or V5 combined with V4R failed to improve sensitivity in Group 1. The monitoring of lead V4R allowed detection of 23% of the ischaemic episodes in Group 2 that would have passed undetected if only lead II and V5 were monitored. These results demonstrate the value of an additional right precordial lead during coronary artery bypass grafting in patients with right-sided occlusive disease.

Key words

Heart; electrocardiography, coronary artery disease, myocardial ischaemia.
Monitoring; electrocardiography.

Peri-operative myocardial ischaemia has recently been established as an independent risk factor in the development of a peri-operative myocardial infarction in patients who have coronary artery bypass surgery.[1,2] Evaluation of the electrocardiographic signals is essential for the anaesthetist to detect myocardial ischaemic events.

The use of a five-electrode ECG system has been generally accepted during all cardiac procedures since the first demonstration of the value of peroperative monitoring of lead V5.[3,4]

Additional monitoring of other precordial leads during anaesthesia was recently shown to improve the sensitivity of ECG monitoring in the detection of peroperative ischaemia.[5] Multiple lead ECGs, however, cannot be used during cardiac surgery because of obvious interference with the surgical field. Nevertheless, in high risk cases it is useful to monitor the right ventricle and the posterior surfaces of the heart. The use of a right-sided precordial lead in the detection of ischaemic events in the right ventricle was evaluated in several cardiological studies.[6-8] The lead V4R was found to have a reasonably high sensitivity, specificity and predictive value for right ventricular ischaemia and infarction. The peroperative use of a V4R was suggested for patients with right-sided coronary occlusive disease,[9] although little is known about its value in the detection of peri-operative ischaemia.

The present study was designed to investigate whether the V4R lead is beneficial in the peroperative monitoring of patients with and without right-sided coronary occlusive disease.

Methods

This study was performed in 60 patients who had elective coronary artery surgery. Thirty-four patients suffered from left-sided coronary artery disease and showed no signs of significant right-sided occlusive lesions (Group 1). The other 26 patients had both left coronary artery disease and significant right-sided occlusive disease on coronary angiography (Group 2).

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Table 1. Clinical characteristics of the patients.

	Group 1	Group 2
Number of patients	34	26
Age, years, mean (SD)	65 (9)	67 (8)
Sex (male/female)	19/15	16/10
Previous medical history		
Previous coronary artery bypass grafting	5%	7%
Previous myocardial infarction	46%	52%
Hypertension	64%	69%
Diabetes mellitus	29%	25%
Medication		
Nitrates	80%	84%
Beta-adrenoceptor blocking drugs	33%	36%
Calcium-channel blocking agents	73%	69%
Other antihypertensive agents	13%	15%
Dipyridamole	70%	73%

Patients with intraventricular conduction abnormalities, pacemakers or an abnormal heart rhythm were not studied.

The clinical characteristics of the patients are shown in Table 1. No significant differences in sex, age, previous medical history and medication was found between the two groups, apart from the different localisation of the occlusive lesions. The electrodes were applied after vigorous skin preparation (alcohol cleansing and dry sponge abrasion). A modified Mason–Likar lead configuration[10] was used with the leg electrodes, placed midway between the anterior and posterior iliac spines. The fifth electrode was placed in the V5 position. A V4 right precordial electrode lead was also applied. This V4R lead consisted of a unipolar precordial exploring electrode, which was placed on the right midclavicular line in the fifth intercostal space. All electrodes and leads were then secured with plastic tape.

A 20-gauge cannula was introduced in the left radial artery under local anaesthesia. Anaesthesia was induced, after pre-oxygenation, with fentanyl 20 μg/kg and diazepam 0.1 mg/kg. Intubation was facilitated with vecuronium 0.1 mg/kg. Anaesthesia was maintained with isoflurane (0.2–0.4%) in air–oxygen mixture (50/50). A flow-guided pulmonary artery catheter was inserted, after intubation, in all patients via the right internal jugular vein. An additional dose of 30 μg/kg fentanyl was administered between this procedure and the start of sternotomy, in a continuous infusion so that each patient received a total dose of 50 μg/kg fentanyl before sternotomy.

ECG leads II, V5 and V4R, and systemic and pulmonary arterial pressures were continuously monitored on an eight-channel recorder (Hewlett-Packard 7758A). High speed recordings were obtained when sympathetic stimulation was likely to occur, such as during arterial cannulation, intubation, pulmonary artery cannulation, skin incision and sternotomy, or when ischaemic ECG changes were seen on the oscilloscope. Myocardial ischaemia was defined as a horizontal or downsloping ST segment depression of 0.1 mV or greater, or an elevation of 0.2 mV or greater, measured 80 msec after the J point, compared to the stable pre-operative and pre-induction tracings. The ECG tracings were interpreted independently by two readers. Discordant readings were not included in the data analysis.

The following causes were sought when myocardial ischaemia was diagnosed: hypotension greater than 20% reduction of mean arterial pressure, compared to awake control value; hypertension greater than 20% increase in mean arterial pressure, compared to awake control value; heart rate elevation of more than 20% of awake preoperative value; ischaemia without haemodynamic changes.

All significant responses in this study were assumed to be true positive responses.[5] The sensitivity of a single lead was calculated as the total number of ischaemic changes detected in that lead/the total number of ischaemic episodes detected in the three leads. The sensitivity of lead combinations was calculated as the total number of ischaemic episodes in which any lead in that combination was positive/the total number of electrocardiographic episodes detected. Unpaired Student's *t*-test was performed where appropriate. Statistical significance was accepted at $p < 0.01$.

Results

Ischaemic episodes were detected in 24 patients (40%). Sixteen of these were accompanied with significant haemodynamic changes, mainly increase in heart rate,[10] and to a lesser degree a significant change in blood pressure (hypertension, 3 and hypotension, 1). The remaining eight ischaemic episodes appeared without the occurrence of significant haemodynamic changes. The incidence of ischaemic episodes was greater in Group 2 (50%) than in Group 1 (32%). However, there was no difference in the incidence of the probable cause between the two subgroups. The mean duration of the ischaemic episodes was 8 (SD 3) minutes (range 2–15 minutes).

No peri-operative myocardial infarction, defined as an infarction becoming apparent during the operation or within one week after the operation, occurred in this group of 60 patients. The sensitivity of the ST segment changes in the different leads and combinations of leads is shown in Figure 1 for the total population, and in Figures 2 and 3 for Groups 1 and 2 respectively. The frequency for the individual leads corresponds to the maximal sensitivity for detection of ischaemia if that lead alone had been monitored, assuming all ischaemic responses were true positives.

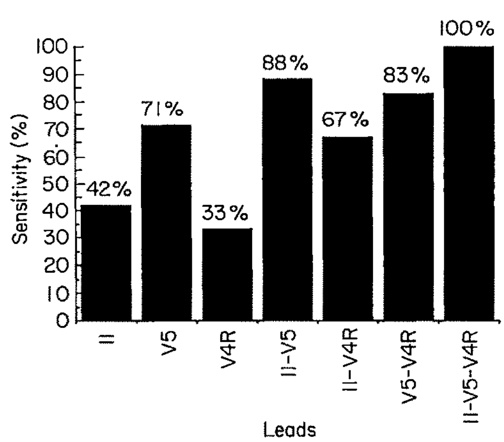

Fig. 1. Sensitivity of the ST segment changes in the leads II, V5 and V4R and in the lead combinations II–V5, II V4R, V5–V4R and II–V5–V4R for the total population studied ($n = 60$). The frequency for the individual leads corresponds to the maximal sensitivity for detection of ischaemia if that lead alone had been monitored.

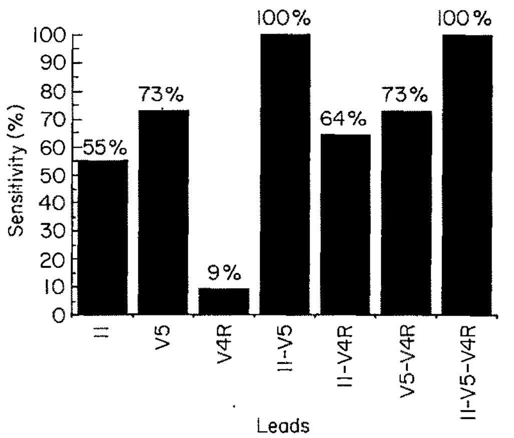

Fig. 2. Sensitivity of ST segment changes in different individual and combined leads, monitored in the group with only left-sided coronary artery disease on coronary angiography (Group 1, 34 subjects and 11 ischaemic episodes, detected on ECG).

Sensitivity of a single lead was greatest for V5 and was comparable in the total population (71%) and the two subgroups (Group 1: 73% and Group 2: 69%). The sensitivity of lead II in the total population was higher (42%) than for the V4R lead (33%). Lead II had a sensitivity of 55% in Group 1 and only of 31% in Group 2. Lead V4R, on the other hand, appeared to be more sensitive in Group 2 (54% versus 9%). For the total population the sensitivity was comparable for the two-lead combination II–V5 (88%) and V5–V4R (83%). The sensitivity of the leads II–V4R combined was only 67%. For Group 1 the standard clinical combination, leads II–V5, allowed 100% detection of the ischaemic epidodes by the three leads monitored in this study. The two-lead combination II–V4R had a sensitivity of only 64%, and the V5–V4R combination was 73% sensitive.

For Group 2, on the other hand, the two-lead combination V5–V4R was highly sensitive (92%). The combination of leads II–V4R had a sensitivity of 69%, while sensitivity of the combination II–V5 was intermediate (77%). For the total population, 13% of the ischaemic changes were detected only in lead V4R and would have remained undetected if this lead had not been monitored. In Group 1 the ischaemic events found in lead V4R were also apparent in lead V5 and (or) lead II. In Group 2, on the other hand, the monitoring of lead V4R allowed identification of 23% ischaemic episodes that would have been missed if only leads II and V5 were monitored.

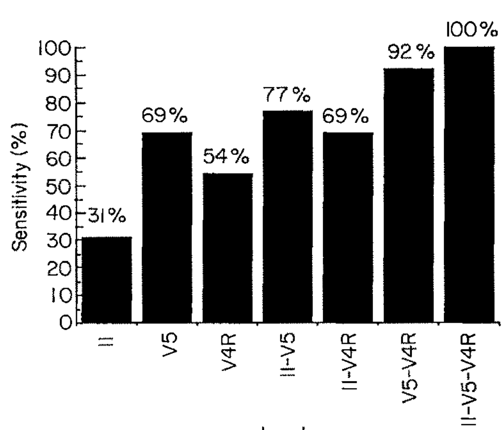

Fig. 3. Sensitivity of the ST segment changes in the different individual and combined leads in the group with combined left- and right-sided coronary artery disease on angiography (Group 2, 26 subjects and 13 ischaemic episodes).

Discussion

Klein and his colleagues proposed the recording of the V4R lead as an intrinsic part of early evaluation and electrocardiographic examination of ischaemic events in the inferior wall and right ventricle, because of its simplicity and high sensitivity (82.7%) and specificity (76.9%). Less is known about the validity of this lead in the monitoring of peroperative ischaemia. Our results demonstrate that the monitoring of a right precordial lead at the V4R position allows the anaesthetist to detect a number of ischaemic events, that otherwise would have passed undetected in patients with right-sided occlusive disease.

We have assumed that all ischaemic changes were true positive responses, with 100% specificity. Therefore, the estimates of sensitivity remain imprecise, since we did not compare these electrocardiographic data with other objective measurements of ischaemia.

Many factors are known to affect the specificity and sensitivity of the electrocardiographic diagnosis of myocardial ischaemia, a number of which are easily encountered during cardiac surgery (hypothermia, hypokalaemia, etc.). Excluding these effects is difficult, given the unstable nature of the peri-operative period. Nevertheless, routine peri-operative diagnosis and treatment of myocardial ischaemia is mainly based on electrocardiographic changes, compared to a rest or control ECG, and in patients with known ischaemic heart disease, ST segment displacement correlates well with ischaemia.[11,12]

The fact that all ischaemic episodes responded well to therapy, either a change of depth of anaesthesia and (or) an infusion with isosorbide dinitrate, provides some confirmation that this was genuine myocardial ischaemia. The degree of ischaemic changes in the V4R lead may also be influenced by ischaemic events that occur in other leads.

ST elevation in the V4R lead, that indicates transmural ischaemia, is a rightward as well as an anteriorly orientated vector. This implies that any ST elevation in the left precordial leads may diminish or even cancel out the ST elevation in the V4R lead and so decrease the sensitivity of this lead for right ventricular ischaemia and infarction.[8] The 32% incidence of ST segment changes found in Group 1 is consistent with the findings of others.[13] However, the high 50% incidence of ST segment changes in Group 2 suggests a more extensive degree of coronary artery disease in this population.

There was no significant difference in duration of ischaemic episodes in the two groups, but it is likely that some kind of bias has occurred, with selection of patients with a more severe degree of coronary artery disease in the second group. It is possible that, since only leads II, V5 and V4R were continuously monitored and included in this study, some of the ischaemic episodes, only apparent in one of the other leads, were missed. This would explain the high sensitivity found for the lead combination II-V5 in Group 1. Moreover, the relatively low incidence of ischaemic episodes in Group 1 is an additional reason to be cautious in quantifying the sensitivity in this subpopulation. The sensitivity of this lead combination in Group 2 is more

consistent with the findings of others.[2]

We had no problems of sterility or interference with surgical access by monitoring the V4R lead, although one can question the presence of an ECG electrode near the surgical field. This could be circumvented by the use of a more lateral right precordial lead.

In conclusion, our results demonstrate the value of an additional right precordial ECG lead in the detection of ST segment changes during coronary artery bypass grafting in the group of patients with significant right-sided coronary occlusive disease.

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Pupillary signs during cardiac surgery

Their use in the prediction of major cerebral deficit following cardiopulmonary bypass

N. M. WOODALL, J. K. MARYNIAK AND A. GILSTON

Summary

Pupil sizes and reactions to light were studied in 100 patients who had cardiopulmonary bypass. Behaviour of the pupils was observed at six stages during the operation. Most patients (71) had pupils of equal size and similar reaction to light at all times. Twenty-three patients developed unequal pupils at some stage, while six had equal pupils throughout but exhibited differing reaction to light. Major cerebral deficit was significantly more common after operation in patients who developed inequality in pupil size than those who did not ($p < 0.05$). No relationship was seen between dissimilar pupil reaction to light and the development of neurological complications. The clinical estimation of pupil size may help to identify those patients who may exhibit postoperative major neurological dysfunction.

Key words

Anaesthesia; cardiac.
Complications; brain injury, death.

Neurological complications of cardiac surgery were recognised since the description by Fox and his colleagues in 1954,[1] and extracorporeal circulation was implicated as a cause of serious neurological morbidity and mortality.[2–4] The detection of peroperative events that produce cerebral damage was attempted using electro-encephalography or its processed forms,[5,6] cerebral blood flow studies,[7,8] and direct intracranial pressure measurements,[9] with varying degrees of success.

Pupillary reflexes are an important component of any neurological examination, and unlike all other neurological signs, remain available for assessment during cardiopulmonary bypass (CPB). We therefore undertook a study of pupil sizes and reactions to light in patients who had cardiac surgery that required CPB. Our aims were to define normal pupillary behaviour patterns, and to establish whether patterns of pupillary behaviour are associated with the development of neurological damage.

Patients and methods

One hundred patients who had cardiac surgery that required CPB were admitted to the study. Patients with pre-existing neurological or eye disease were excluded. Premedication with lorazepam, papaveretum and hyoscine, was followed by induction of anaesthesia with intravenous barbiturates or fentanyl, maintained with nitrous oxide and oxygen, and supplemented with opiates, volatile agents and muscle relaxants as clinically indicated. Hypothermic CPB was started after systemic heparinisation. The heart was vented in all procedures during which a chamber was opened, and in some closed procedures according to surgical preference. All patients were sedated with papaveretum and midazolam after surgery, and electively ventilated overnight.

Behaviour of the pupils was observed at six stages of the procedure as shown in Table 1. The size of each pupil was assessed, at each of these stages, to the nearest 0.5 mm with the pocket gauge described by Gilston.[10] Reaction to light was assessed by direct illumination of the pupil using a bright laryngoscope. The patients were placed into one of the categories shown in Table 2, according to pupillary size and reaction to light. Equality in size and similar reaction to light were considered normal behaviour (Groups A, B, and C). Any patient who developed unequal pupils or differing reaction to light at any stage was assigned to the

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Table 1. Stages of pupil observation.

1.	After induction of anaesthesia.
2.	At heparinisation.
3.	At maximal hypothermia on bypass.
4.	Immediately before rewarming.
5.	Before coming of bypass.
6.	20 minutes after coming off bypass.

Table 2. Categories of pupil behaviour.

Group	Size	Reaction to light
A	Always equal	Always reacting
B	Always equal	Always nonreacting
C	Always equal	Variable reaction
D	Always equal	Different reaction
E	Unequal	Similar reaction
F	Unequal	Different reaction

appropriate abnormal group (D, E or F). Since anisocoria (inequality in pupil diameter) of 0.4 mm is not abnormal,[11] we assigned patients to an unequal pupil category only if the difference in size was 1 mm or greater.

Heart rate, pump flow, and mean arterial pressure were recorded, as was the administration of opiates, atropine and vasoactive drugs. The interval between withdrawal of CPB and response to commands and movement of all four limbs was noted. The day after surgery, all patients were examined for the presence of major neurological disorder, principally confusion, disorientation, or gross motor dysfunction.

Statistical methods

Multiple regression analysis and Chi-square tests with Yates' correction were used as appropriate to analyse the data. $p < 0.05$ was considered statistically significant.

Results

The study group consisted of 76 males and 24 females with a mean age of 53 years (range 9 to 77). Fifty-nine patients underwent coronary artery bypass grafting. The remaining 41 underwent open procedures. Thirty-three had one or more valves replaced, five had correction of congenital cardiac defects, two had repair of ventricular septal defect, and one patient had an ascending aortic aneurysm resection.

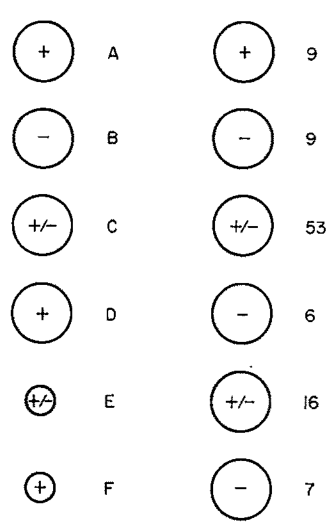

Fig. 1. Number of patients in each category of pupil behaviour.

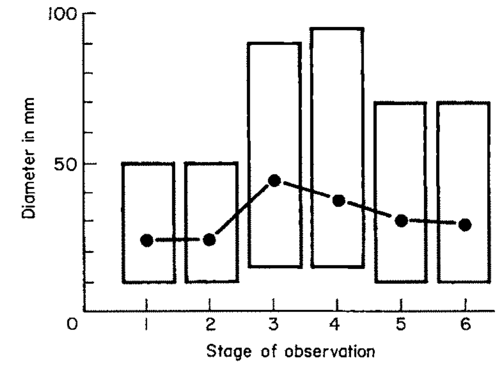

Fig. 2. Pupillary sizes, mean and range at each of the six observation stages.

Pupillary behaviour

Seventy-one patients fell into categories A, B or C, and had pupils of equal size and similar reaction to light at all times (Fig. 1). The majority of patients (53) fell into Group C; their pupils reacted on some occasions and not others. Reaction to light in nine patients (Group A), was present throughout, while in a further nine, no reaction was seen at any time.

Twenty-nine patients exhibited abnormal pupillary behaviour (Groups D, E and F). Six patients (Group D) had pupils of equal size throughout but showed differing light reactions. The remaining 23 had pupils of unequal size at some stage; 16 of these (Group E) reacted similarly to light, seven (Group F) did not.

The changes in size and reaction to light in all 100 patients are summarised in Figures 2 and 3. Mean pupil size was equal at stages 1 and 2 (mean 2.4 mm, range 1–5 mm) and increased to maximum diameter (mean 4.4 mm, range 1.5–9 mm) after institution of CPB and hypothermia (stage 3). Pupil size returned towards prebypass values (mean 2.9 mm, range 1–7 mm at stage 6) after rewarming and weaning from bypass.

Sixty-five percent of pupils reacted to light after induction of anaesthesia (stage 1), but showed decreased reacti-

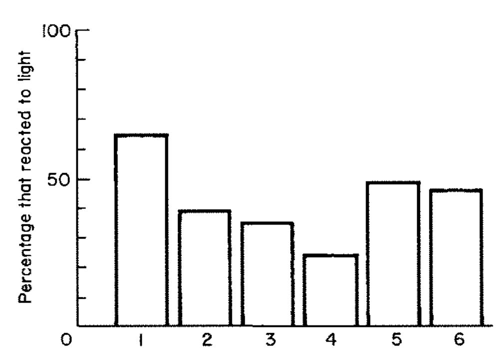

Fig. 3. Pupillary reaction to light at each of the six observation stages.

Table 3. Relationship between anisocoria, neurological morbidity and overall mortality.

	Number of patients	Neurological dysfunction	Death
Isocoria	77	2*	0**
Anisocoria	23	4*	3**
Total	100	6	3

Differences between groups: *$p < 0.05$, **$p < 0.02$.

vity after further opiate administration (39% reacting at stage 2) and institution of CPB (35% at stage 3). Pupillary reactivity increased after rewarming (49% at stage 5).

Neurological complications

The incidence of neurological complications and their relationship to pupillary behaviour is summarised in Table 3. Six patients fulfilled the criteria for major neurological complications after operation. Four of these had anisocoria, three of whom subsequently died.

Patient 1 was a 59-year-old, 107-kg, hypertensive diabetic woman who underwent repair of an ascending aortic dissection with a dacron graft. She was fully conscious and neurologically intact before operation. She regained consciousness the morning after surgery with a dense left-sided hemiplegia. Weaning from positive pressure ventilation failed and she died 10 days later. Her pupils were unequal (left 5 mm, right 6 mm) before she came off bypass.

Patient 2 was a 62-year-old man admitted for coronary artery surgery. He was extubated on the first day after operation, following which he was noted to be disorientated and suffered two generalised convulsions. A neurological opinion indicated brainstem infarction. His neurological function further deteriorated and he died 3 weeks later. His pupils were unequal (right 4 mm, left 3 mm) during hypothermia (stage 3).

Patient 3 was a 63-year-old 100-kg man who underwent elective coronary artery bypass grafting. Surgery was complicated by prolonged and difficult weaning from CPB. He regained consciousness the next morning but had developed a left-sided hemiplegia. IPPV was continued for a further 5 days until he died of circulatory failure. Inequality in pupil size was noted at stage 6 (right 3 mm, left 2 mm).

Patient 4 was a 55-year-old 89-kg diabetic man who underwent coronary artery surgery. Pupillary inequality was noted at maximal cooling on bypass (stage 3) during an otherwise uneventful anaesthetic and operation. He woke up 6 hours after weaning from bypass, but was aggressive and displayed inappropriate behaviour, although he showed no other gross neurological deficit. He made a complete recovery.

Two further patients exhibited confusion and disorientation during the first day after operation, but made a full recovery. No anisocoria was noted.

Twenty-three of the 100 patients studied developed anisocoria. Four of the six patients who showed signs of gross postoperative neurological dysfunction (including those who died) were in this category. The incidence of postoperative neurological damage was significantly greater in those patients who exhibited anisocoria peroperatively than those who did not ($p < 0.05$). The three patients who died with severe neurological complications all came from the anisocoric group. This distribution is also significant ($p < 0.02$). In all of the six patients described above both pupils responded similarly to light at all observation stages.

Multiple regression analysis of demographic and haemodynamic data, drug administration, pump flow per unit surface area, wake up times, and abnormal pupillary behaviour revealed a significant positive correlation only between prolongation in time taken to wake up and advancing age ($p < 0.001$).

Discussion

Examination of the pupils is an established component of both neurological assessment and anaesthetic monitoring. However, there are few published data on the behaviour of the pupils of the anaesthetised patient during CPB. This is surprising in view of the recognised association between cardiac surgery that requires CPB and postoperative neurological deficit. Our study of 100 patients describes patterns of pupillary behaviour during cardiac surgery. Maximal dilatation occurred at minimum temperature on bypass (20% of patients had pupils of 6 mm diameter or greater), and returned to prebypass levels towards the end of surgery. Absolute pupil size and differing reaction to light bore no relationship to postoperative neurological complications. However, we found a significant association between anisocoria and neurological deficit after CPB.

Reported incidence of cerebral damage has varied widely from 0 to 100%,[3,4,12,13] depending mainly on the depth and timing of postoperative assessment and other differences in study design. We deliberately employed simple and universally available methods to assess pupil size and postoperative neurological status. Our detection rate for complications might have been higher if we had used more sophisticated techniques. However, we were able to identify a specific group of patients in whom cerebral damage is more likely to have occurred during surgery, and is associated with an increased postoperative mortality.

Acknowledgments

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Multidimensionality of psychological recovery from anaesthesia

Analysing six recovery tests

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Summary

Six recovery tests, choice reaction time (CRT), CRT doubletask, finger tapping test (FTT), critical flicker fusion frequency (CFF), Maddox wing, and p-deletion test, which cover basic cognitive, motor and perceptive functions as well as concentration, were analysed and compared with each other. Correlation between these tests after recovery from standardised general anaesthesia was calculated in 22 patients. Moderate to high correlation was found between CRT and CRT doubletask ($r = 0.62$ to $r = 0.73$), when parameters of six different tests were compared. Finger tapping correlated moderately with the movement time of both the CRT and the CRT doubletask ($r = 0.46$ and $r = 0.47$ respectively). We concluded that the CRT test, which measures initiation and movement time, might replace the CRT doubletask and the FTT. The CFF correlated moderately with the initiation time of the CRT doubletask, but because a slightly different function seems to be involved, further research is needed. Maddox wing and the p-deletion test correlated with no other test. Results indicated that recovery is differentiated in at least four distinct psychomotor functions, which should be tested by CRT (to measure initiation and movement time), Maddox wing and p-deletion.

Key words

Anaesthesia; outpatient; recovery.
Psychomotor tests; choice reaction time; finger tapping test; critical flicker fusion frequency; Maddox wing; p-deletion test.

Rapid recovery from general anaesthesia is important in daycase surgery to permit early discharge of patients. Consequently, anaesthetists have become aware of the relevance of evaluation of recovery by psychological performance testing.[1] A wide variety of tests were applied, from the patient's ability to open eyes[2] to the use of advanced computerised test methods.[3] However, tests to evaluate recovery have not been standardised and many investigators use a test battery which is not relevant to all aspects of recovery. Furthermore, the majority of the tests measure several psychological functions at the same time, such as competence, ability and motivation without specifying the relative contribution of each to the results.

The purpose of this study was to assess and evaluate correlations between six tests that measure 11 test parameters, which include the major functions relevant to recovery to see if different functions have an independent rate of recovery. An attempt was made on the basis of these data to determine the minimum requirements for a test battery for recovery from anaesthesia.

Patients and methods

Twenty-two ASA class 1 patients aged 18 to 57 years, scheduled for diagnostic arthroscopy of the knee in the daycase surgery unit participated. The study was approved by the Medical Ethics Committee of the hospital and all patients gave verbal informed consent. The patients were examined before operation. No medication likely to affect recovery from general anaesthesia was taken by any of the patients and no premedication was given.

After pre-oxygenation, anaesthesia was induced with 1 mg/kg methohexitone, followed by atracurium 0.5 mg/kg. Anaesthesia was maintained, after tracheal intubation, with 66% nitrous oxide in oxygen using a circle system and absorber with a total fresh gas flow of 3 litres/minute, and patients' lungs were ventilated to maintain end-expiratory CO_2 between 4 and 5%. Isoflurane 3% (inspired) was given before intubation and 0.9% (inspired) during maintenance. The isoflurane and nitrous oxide were stopped at the end of surgery which was defined as the end of anaesthesia. Neuromuscular blockade was reversed with atropine and neostigmine. No other drugs were given before or during anaesthesia.

Assessment of recovery

The tests that follow (total of 11 variables) were performed before operation to set a baseline for each patient, after a trial to exclude learning effects, and after operation at 30, 60, 90 and 120 minutes from the end of anaesthesia.

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CRT test. In the CRT test, after release of a start button one of five possible stimuli (lighting buttons) must be pressed as fast as possible after presentation. These stimuli are presented randomly in a series of 20, with undetermined intervals of 50 to 999 msec. Several processes can be differentiated within the reaction,[4] of which the choice reaction time test subsequently estimates the *initiation time* (a cognitive component of the reaction time, measured by the release of the initially held button) and the *movement time* (a motor component of the reaction time, measured by the movement from the initially held button to the stimulus button). Initiation time and movement time together make up the *total reaction time.* Initiation time, movement time and total reaction time are expressed in msec.

CRT doubletask. This test, like the CRT test, measures reaction time but the patient also has to count the appearing stimuli. One out of the five possible stimuli buttons must be excluded; at each subsequent presentation of the CRT doubletask, a different stimulus button must be excluded to prevent recognition. The patient's attention is divided, and the reaction time is slowed down as a result of the simultaneous counting and reacting. The order of the stimuli and the interstimulus intervals are again randomly defined, as with the CRT test. Initiation time, movement time and total reaction time are measured in msec.

FTT. This is also called the tapping test[5] and is a simple assessment of manual dexterity to estimate motor ability.[6] A button has to be pressed as many times as possible within 10 seconds. This test is conducted twice at each presentation, and the results are presented as mean interval time between button presses in msec.

CFF test. This is a visual perception test to assess cortical arousal sensitive to the effects of psychotropic drugs.[7] A flickering visual stimulus with increasing frequency is presented to the patient, and when the subjective impression of flickering changes into that of constant shining, a button must be pressed. The test is then repeated in reverse order: the visual stimulus is presented with a high frequency that decreases until the subjective impression of flickering appears. The button must be pressed again at that moment. The results are presented in mean frequency at the time of button-press in hertz.

Maddox wing. This measures ocular imbalance, caused by incoordination of the extra-ocular muscles.[8] The test consists of a viewer which divides the field of vision of both eyes, so that the patient sees an arrow with the right eye, and a numbered scale with the left. Divergence of the eyes causes the arrow apparently to move along the scale, and the subject is asked to what number the image of the arrow points when it has ceased moving. Eye divergence was measured in prism diopters.

P-deletion test. This is a letter-cancellation task as described by Dixon and Thornton.[9] Every patient received a text that contained 140 ps and was asked to delete as many as possible in 3 minutes. The number deleted measured perceptual speed, and sustained attention. The number missed measured the accuracy of the patient.

In addition, a questionnaire was sent to every patient 4 weeks after surgery, to ask their opinion about recovery and detect possible discrepancies between recovery measured by tests and patient opinion.[10]

Table 1. Patient age, sex ratio, weight and duration of anaesthesia (mean, SD) ($n = 22$).

Sex ratio (F/M)	11/11
Age (years)	29.7 (10.7)
Weight (kg)	69.9 (12.4)
Duration (minutes)	47.7 (10.9)

Statistics

We compared tests at 120 minutes after operation to optimise differences. Differences between tests were maximal at this time, but no major differences in correlations between tests were found at subsequent times during recovery. Differential scores for each test were computed by subtracting the pre-operative score of each patient from the scores at 120 minutes after operation. Consequently, using differential scores, the variance due to differences in individual scoring levels was excluded. All subsequent analyses were performed using these transformed data.

Only standardised tests with proven reliability in neuropsychological testing were used[11] except for the CRT doubletask (a complex reaction-time task) which was modified. To control random variance scores which can contribute to unreliability of results, a test-retest correlation coefficient was computed for all the measures taken. By using the differential scores at 90 and 120 minutes after operation, individual differences in the recovery of psychomotor functions were minimal, so that a test-retest correlation coefficient (Spearman rank correlation coefficient) indicated the random variance of the test scores. Recovery enhances the variance of the scores between two trials, so this test-retest correlation gives an overestimation of the random variance of these tests. Rank scores were used, which indicated the order of recovery of the patients for each test. Tests-retest correlations were considered to be moderate if the correlation coefficient was above 0.40 ($p < 0.05$) and high if it was above 0.70 ($p < 0.01$).

Comparison between the tests was performed by correlation of the patients' rank scores, to compare the relative order of recovery of patients on each test. Again, the Spearman rank correlation coefficient was used. Moderate ($r \geq 0.40$) to high correlation ($r \geq 0.70$) in the order of patient recovery in tests that measure the same psychomotor function (i.e. simple motor ability) indicates that probably the same psychomotor function is being measured by both tests. A low correlation ($r < 0.40$) between the order of recovery of patients with different tests measuring different psychomotor functions, indicates that recovery of these different functions is independent.

Results

Age, weight and duration of anaesthesia for all patients are listed in Table 1. Cardiovascular variables remained stable throughout the whole procedure. All patients underwent anaesthesia and arthroscopy without complications. All patients needed reversal of neuromuscular blockade at the end of anaesthesia.

Scores (mean, SEM) on each test are presented in Table 2, to give the absolute scores after operation at 30, 60, 90 and 120 minutes, and the differential scores at 90 and 120 minutes. Table 3 gives the test-retest correlation coefficients of the various tests between differential scores at 90 and 120 minutes postoperatively, which varied from 0.63 to

Table 2. Pre-operative scores; scores at 30, 60, 90 and 120 minutes after operation, and differential scores at 90 and 120 minutes, mean (SEM).

	Before anaesthesia	After operation (minutes)				Differential scores (minutes after operation)	
		30	60	90	120	90	120
CRT							
Initiation time	411 (8.3)	509 (28.2)	446 (11.4)	435 (10.5)	430 (11.8)	30 (5.5)	23 (9.9)
Movement time	143 (7.1)	203 (16.3)	173 (10.4)	166 (9.1)	155 (9.2)	24 (5.6)	12 (6.6)
Total reaction time	554 (11.5)	713 (41.1)	620 (17.4)	601 (14.8)	585 (16.3)	54 (8.1)	35 (13.6)
CRT double task							
Initiation time	468 (9.3)	516 (17.0)	480 (12.7)	467 (12.4)	454 (10.4)	6.3 (9.7)	−8 (8.1)
Movement time	165 (9.8)	205 (13.9)	188 (12.2)	176 (10.3)	174 (11.1)	13 (8.1)	11 (9.5)
Total reaction time	633 (13.5)	720 (22.9)	668 (18.1)	644 (17.8)	628 (16.9)	19 (15.1)	3 (15.6)
FTT	166 (6.2)	188 (7.1)	172 (5.3)	170 (4.6)	166 (5.5)	4 (3.5)	1 (4.3)
CFF	32.4 (1.0)	28.0 (0.9)	29.8 (1.0)	30.6 (0.9)	31.4 (0.9)	−2.0 (0.6)	−1.2 (0.5)
MW	2.5 (0.5)	7.9 (0.8)	5.0 (0.5)	3.9 (0.6)	3.3 (0.5)	0.8 (0.3)	0.8 (0.3)
p deletion	58 (3.0)	41 (2.4)	53 (2.5)	58 (3.2)	60 (3.2)	1 (2.2)	2 (2.3)
Missed ps	11 (3.0)	10 (3.1)	10 (2.7)	10 (3.0)	11 (3.0)	−1 (1.6)	−1 (1.4)

CRT, choice reaction time; FTT, finger tapping test; CFF, critical flicker fusion frequency; MW, Maddox wing.

Table 3. Test-retest correlation (90–120 minutes) (Spearman Rank correlation coefficient) ($n = 22$).

CRT test	
Initiation time	0.63**
Movement time	0.82**
Total reaction time	0.81**
CRT double task	
Initiation time	0.70**
Movement time	0.90**
Total reaction time	0.90**
Finger tapping test	0.79**
CFF	0.70**
Maddox wing	0.73**
p-deletion test	0.80**
Missed ps	0.83**

CRT, choice reaction time; CFF, critical flicker fusion frequency.
**$p < 0.01$.

0.90 ($p < 0.01$). All test-retest correlations were high, except for the initiation time of the CRT which has a modest test-retest correlation.

Full recovery, as measured by the opinion questionnaire took a mean time of 1.5 days. The occurrence of differential recovery in different psychological functions is shown by differences in the sequence of recovery in various tests to demonstrate differential recovery. Correlations between the relative recovery of patients in each test at 120 minutes postoperatively are presented in Table 4. There was a moderate correlation between the initiation time of both the CRT test and the CRT doubletask tests ($r = 0.62$), a high correlation between movement time ($r = 0.73$) and low correlations between initiation time and movement time ($r = 0.32$ and $r = 0.36$). The finger tapping test correlated moderately with the movement time of both the CRT and the CRT doubletask ($r = 0.46$ and $r = 0.47$ respectively). Correlations with all other tests were low ($r < 0.40$). The CFF test correlated moderately with the initiation time of the CRT doubletask ($r = 0.57$), which was also reflected in a moderate correlation with the total reaction time of the CRT doubletask ($r = 0.40$). Correlations with all other tests were low ($r < 0.40$).

Both the Maddox wing and p-deletion test (the number of ps deleted and the number of ps missed) correlated low to very low with all other tests.

Discussion

The choice of psychomotor and cognitive recovery tests after anaesthesia is an important but difficult issue. Recov-

Table 4. Differentiation between factors within recovery: correlation between tests (Spearman Rank correlation), 120 minutes after operation ($n = 22$).

	CRT Initiation time	CRT Movement time	CRT Total reaction time	CRT-double task Initiation time	CRT-double task Movement time	CRT-double task Total reaction time	FTT	CFF	MW	p deletion
CRT										
Movement time	0.01									
Total reaction time	0.81**	0.50								
CRT double task										
Initiation time	0.62**	0.32	0.57**							
Movement time	0.36*	0.73**	0.64**	0.45*						
Total reaction time	0.54**	0.56**	0.68**	0.81**	0.85**					
FTT	0.25	0.46*	0.37*	0.18	0.47*	0.31				
CFF	−0.31	−0.33	−0.38*	−0.57**	−0.21	−0.40*	−0.09			
Maddox wing	0.33	−0.11	0.26	0.12	−0.27	−0.08	−0.02	−0.24		
p deletion	−0.18	0.04	0.03	−0.14	−0.15	−0.23	−0.21	0.17	−0.03	
Missed ps	0.36	−0.00	0.25	0.10	−0.02	0.02	0.13	−0.24	0.13	−0.18

*$p < 0.05$, **$p < 0.01$.
CRT, choice reaction time; FTT, finger tapping test; CFF, critical flicker fusion frequency; MW, Maddox wing.

ery tests should be complete, reliable and valid, to permit conclusions to be drawn from their results, and cover all aspects, otherwise patients may be sent home too soon. Different test situations measure different aspects of recovery, but in all cases basic cognitive and psychomotor functions must be assessed.

Recovery tests should be as simple as possible, practical, and cause the patient minimal strain. Measuring identical parameters should be avoided. For completeness and simplicity, we compared six tests that measured 11 different test variables and covered basic cognitive and psychomotor functions. The problem with tests which measure directly functions important for the patient to return home, such as walking and driving, is that there are too many to measure. Furthermore, these tests are often insensitive to small changes in performance, and heavily depend on prior level of mastery, and the patient can be motivated to perform better than in a real life situation.[1] We therefore chose to test basic functions which cover the relevant aspects of recovery.

We selected six commonly used tests, described by Kortilla,[12] which comprise some basic functions: perception, coordination, motor function, and different aspects of cognition. We investigated the sequence of recovery of patients in different tests to see if different aspects of recovery were assessed, and to ascertain whether there was multiple testing of the same function. The results show medium to high correlation between comparable functions measured by the CRT and the CRT doubletask. This indicates that initiation time and movement time are separate elements of recovery, measured by both the CRT and the CRT doubletask. Furthermore, these results give an indication of the correlation that can be expected between tests that are closely related.

Movement time in both tests correlated moderately with the finger tapping test; both measured the motor ability of the patient, but finger tapping correlated low to very low with initiation time. The CFF test correlated moderately with the initiation time of the CRT doubletask, and probably indicated that cognitive reaction forms part of the functions measured in this test. The Maddox wing and p-deletion test showed no correlation with the above-mentioned tests, because these tests apparently measure different functions in the recovery of the patient. The correlation between the number of ps deleted and the number missed was very low and indicated that different functions are involved. The very low variance in the number of ps missed within each patient indicates the low influence of recovery.

To test economically and cause as little discomfort to the patient as possible, a choice has to be taken between tests which apparently measure the same function. The CRT test was more basic, more general in use and included measurement of both initiation time and movement time so we preferred it to the CRT doubletask and the finger tapping test. The CFF test correlated moderately with the initiation time of the CRT doubletask, but these tests cannot be said to measure the same function on the results of this study alone; more studies are needed to confirm connexion between functions measured by the CFF and the CRT doubletask.

Maddox wing and p deletion showed no correlation with the above mentioned tests, and indicated that these probably measure distinct psychomotor functions. To cover these functions (perceptual accuracy, perceptual speed and sustained attention), these tests should be included in the measurement of recovery. The number of ps missed seem to give no indication of recovery and could be omitted.

The tests in this article measured several psychomotor functions, and indicated that recovery is not a unidimensional concept. This implies that several tests are necessary to measure street-fitness, home-readiness or full recovery.[12] We conclude, after comparison of 11 variables of six frequently used tests that cover major aspects of recovery, that at least four functions measured by three tests can be distinguished, as can be seen by differences in recovery rate for these tests which cannot be attributed to differences in sensitivity of the tests. To include all aspects of recovery important in the decision to send patients home, motor ability and coordination as well as perception and cognitive recovery need to be tested. The choice reaction time, the Maddox wing and the p-deletion tests could fulfil this need.

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Continuous versus intermittent epidural analgesia

A randomised trial to observe obstetric outcome

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Summary

A randomised study of 381 women was carried out to compare the obstetric outcome after epidural analgesia maintained by an intermittent top-up regimen or with a continuous infusion. The two groups were well matched with respect to age, parity, mode of onset of labour and indication for epidural. Maintenance of epidural analgesia by continuous infusion resulted in a significantly decreased need for top-up doses. A reduction in the incidence of hypotension, cardiotocographic evidence of intrapartum fetal hypoxia and Caesarean section was associated with this. It is concluded that the maintenance of epidural analgesia by continuous infusion is a safe and reliable method and may be more advantageous and less labour intensive than the traditional intermittent regimen.

Key words

Anaesthetic techniques, regional; epidural.
Surgery; obstetric.

The instillation of a local anaesthetic into the epidural space has become established as a safe and effective method of pain relief during labour.[1] The use of a continuous epidural infusion has been introduced in recent years to try to overcome some of the disadvantages of the traditional intermittent regimen.[2] The main purpose of these studies has been to establish the optimum concentration of local anaesthetic and infusion rate, but there has been little reference to obstetric complications or benefits. We have set up a randomised trial to compare the obstetric and neonatal outcome after a traditional intermittent regimen with that of a standard continuous infusion.

Methods

Three hundred and eighty-one women were included in the study between December 1986 and November 1987, after written consent was obtained. All the mothers were in labour at term and carried singleton, normally formed babies, who presented cephalically and with no factors to contraindicate the use of epidural analgesia. The indication for epidural was either patient request or medical recommendation for conditions such as intrapartum hypertension. The mothers were divided into two groups, using computerised random number tables, either an intermittent group (193) or a continuous group (188). The randomisation was stratified so that the multiparae and primiparae were randomised separately.

Anaesthetic protocol

A standard technique was employed to introduce the epidural catheter. A test dose of 3 ml 0.5% bupivacaine was injected when the anaesthetist was satisfied that the catheter was correctly situated and the mother observed for 20 minutes. The loading dose was completed by a further injection of 10 ml 0.25% bupivacaine provided the anaesthetist was satisfied. An envelope was selected at this stage that contained a randomly allocated method for the continuation of analgesia. A bolus of 10 ml 0.25% bupivacaine was administered when more analgesia was requested by the patient if the intermittent method was chosen. An infusion of 0.125% bupivacaine was started at 10 ml/hour if the continuous method was chosen. Ten millilitres of 0.25% bupivacaine could be given as a bolus if the analgesia was inadequate, but if this was still inadequate the anaesthetist was informed. The fetal cardiotocograph (CTG) and maternal blood pressure, heart rate, respiration and level of consciousness were checked carefully at the time of injection or top-up.

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Obstetric protocol

Standard obstetric and midwifery practices were applied to all mothers, independent of epidural method, until the cervices were found to be fully dilated. The following regimen was followed at full dilatation, provided the CTG was normal:

Intermittent group. The mother was encouraged to push, if she had the urge to bear down but if not, no action was taken. A top-up was given if she was in pain. The mother was encouraged to bear down whenever she developed the urge to do so, or after 3 hours of full dilatation, or earlier if the CTG became abnormal.

Continuous group. The infusion was stopped if the mother had the urge to bear down and she was encouraged to do so, but if not the infusion was continued for a further 2 hours and then stopped. One hour later (up to 3 hours after detection of full dilatation) the mother was urged to bear down. At any stage after full dilatation, if the CTG became abnormal, or if the mother felt the urge to bear down she was encouraged to do so.

A time limit of 3 hours for the passive phase of the second stage was therefore placed on both groups, at which time the woman was encouraged to push whether or not she had the urge to do so. The management of the expulsive phase of the second stage remained as it is normally practised for both groups. The degree of cervical dilatation at, or nearest the time of insertion of epidural was recorded.

Hypotension was defined as an absolute decrease in the systolic arterial blood pressure to 100 mmHg or lower, or alternatively as a relative decrease of 30 mmHg in the systolic pressure. The cardiotocograph was considered abnormal if there was a change in the CTG to a persistent bradycardia, tachycardia, loss of baseline variability or recurrent decelerations. Care was taken at all times to avoid supine hypotension, and if the woman was not comfortable on her left side a cushion or pillow was used to give left lateral tilt.

Data were analysed using the SPSSX package and Minitab version 5.1. Variables to record the time of various stages were $\log_{10}$ transformed in order to reduce the skewed distribution observed in the raw data. Statistical significance was tested using Chi-square and the Student's t-test; the Mann–Whitney test was applied to Apgar scores and number of top-ups required.

Results

The mothers' comparability is shown in Table 1. There was no difference between the groups with respect to age, parity, mode of onset of labour or indication for epidural. The cervices of those women in the intermittent group were more dilated at insertion of epidural. The obstetric outcome is shown in Table 2. There was no difference between the groups with respect to the length of first stage even after allowance was made for the difference observed for cervical dilatation, length of active (expulsive phase) of second stage, length of third stage or insertion-to-delivery interval.

Significantly more top-ups were needed where analgesia was maintained by intermittent boluses, as would be expected; top-ups required for instrumental delivery are included in these figures (Table 3). More mothers required one or more top-ups in the intermittent group. There was no difference between the groups with respect to the mode of delivery although there was a trend towards an increase in the rate of Caesarean section in the intermittent group. Hypotension was more common in the intermittent group and this may reflect the significantly greater number of top-ups in that group. The significant increase in the incidence of abnormal CTGs may in turn reflect the increased incidence of hypotension (Table 2).

The neonatal outcome is shown in Table 4. There was no difference between the groups with respect to Apgar scores at one and 5 minutes or the need for intubation for resuscitation. The rate of admissions to the special care

Table 1. Comparability of the two groups.

Variable	Intermittent group (n=193)	Continuous group (n=188)
Mean age, years (SD)	25.8 (5.5)	25.7 (5.3)
Range	15–40	16–40
Parity (%)		
Primiparae	83	87
Multiparae	17	13
Onset of labour		
Spontaneous	80	73
Induced	20	27
Indication for epidural		
Patient request	66	64
Medical recommendation	34	36
Mean cervical dilatation at insertion, cm (SD)	4.6 (2.0)	3.9 (1.8)*

*(t=3.84; $p<0.001$).

Table 2. Obstetric outcome.

Variable	Intermittent group (n=193)	Continuous group (n=188)
Mean length of 1st stage, hours (SD)	11.5 (1.6)	11.9 (1.6)
Mean length of 2nd stage, hours (SD)	1.7 (2.3)	1.5 (1.1)
Mean length of active 2nd stage, minutes (SD)	37.0 (2.4)	45.0 (2.0)
Mean length of 3rd stage, minutes (SD)	5.0 (1.4)	5.0 (1.5)
Mean insertion-delivery interval, hours (SD)	6.1 (1.9)	6.8 (1.8)
Number of top-ups required (SD)	1.63 (0.8)	1.14 (0.7)*
Number who required ⩾1 top-up	109.0 (58%)	78.0 (41%)**
Mode of delivery (%)		
Spontaneous vertex	55	56
Low cavity instrumental	20	24
Rotational instrumental	9	8
Caesarean section	16	12
Hypotensive episodes	16	11
Abnormal CTG	32	19

Active 2nd stage=duration of maternal expulsive efforts; CTG, cardiotocograph.
*$p<0.001$.
**Chi-squared=7.97; $p<0.005$.

Table 3. Number of top-ups in each group (percentage in parenthesis).

Number of top-ups	Continuous	Intermittent
0	52 (27.7)	38 (19.7)
1	58 (30.9)	46 (23.8)
2	41 (21.8)	41 (21.2)
3	24 (12.8)	32 (16.6)
4	8 (4.3)	16 (8.3)
5	4 (2.1)	12 (6.2)
6	1 (0.5)	4 (2.1)
7	0	2 (1.0)
8	0	1 (0.5)
9	0	1 (0.5)

baby unit was almost three times more common in the intermittent group although this did not reach statistical significance. Although not part of the study, the quality of pain relief appeared superior in the continuous group in those in whom it was assessed (Table 5).

Discussion

Epidural analgesia is a safe and reliable method for the induction of pain relief in labour. In 1987 in the Princess Anne Hospital, Southampton, epidural analgesia was used by 23% of women in labour, 85% of whom were primiparous. The traditional intermittent regimen is used in most labour wards. This gives intermittent pain relief by its very nature and is associated with episodes of hypotension, which may cause cardiotocographic changes suggestive of intra-uterine fetal hypoxia. As a result of this, midwives must be even more attentive, and vital recordings are carried out every 5 minutes for 20 minutes after each top-up, a system which is very labour intensive.

We believe this present study to be the largest randomised study to observe obstetric outcome and no bias could be found between the groups with respect to age, parity, mode of onset of labour or indications for an epidural. Cervimetrically, those women in the intermittent group were more advanced in labour at insertion, but the insertion-to-delivery interval was similar in both groups. This may be an unreliable comparison since cervical dilatation is an estimate rather than a finite measurement and the recordings were not made at a fixed time in relation to the onset of analgesia. This suggests, if the comparison is reliable, that either continuous epidural speeds up labour or the intermittent regimen can cause prolongation of labour. Bogod *et al.*[3] found that the first stage of labour was significantly prolonged by an intermittent regimen. We know that if hypovolaemia and hypotension are avoided

Table 4. Neonatal outcome.

Variable	Intermittent group ($n=193$)	Continuous group ($n=188$)
Median Apgar Scores		
1 minute	7	7
5 minutes	8	8
Intubation for resuscitation	13 (6.7%)	18 (9.6%)
Admitted to Special Care Baby Unit from Labour Ward	9 (4.7%)	3 (1.6%)

Table 5. Patient acceptability.

	Intermittent	Continuous
Pain relief first stage		
Fully satisfied (%)	58 (29)	71 (36)
Helped (%)	40 (20)	24 (12)
No benefit (%)	2 (1)	5 (3)
Pain relief second stage		
Fully satisfied (%)	40 (19)	70 (34)
Helped (%)	46 (22)	24 (12)
No benefit (%)	14 (7)	5 (3)
Time from delivery to		
Spontaneous micturation (hours)	3.5 (2.6)	5.1 (4.9)
Sensation return (hours)	3.7 (2.7)	4.6 (2.7)
Motor return (hours)	5.0 (3.1)	5.8 (3.0)
Catheterisation after delivery	5 (3)	6 (4)

during the first stage, epidurals do not cause prolongation of labour[4] so this observed prolongation may be due to the increased number of hypotensive episodes in the intermittent group.

There would appear to be no harm done by the use of the continuous regimen, with respect to obstetric outcome, since there was no difference between the groups in length of labour or mode of delivery. The spontaneous delivery rate of approximately 55% in our study was greater than the rate (35%) quoted in other studies.[3,5] This may be because we allowed a passive phase of second stage of up to 3 hours in each group. This was to try to reduce the use of rotational forceps found by Studd *et al.*[6] and followed the study of Kadar *et al.*[7] who showed that patients without the urge to bear down can be left for up to 3 hours in the second stage, and this would lower the instrumental delivery rate. They also found no difference in the proportion of rotational forceps with duration of second stage. Kadar's study referred to intermittent epidurals but found that after 3 hours the subsequent spontaneous delivery rate was less than 30% depending on maternal age and birthweight. The Caesarean section rate of 11% is much lower than that quoted by Bogod *et al.*[3] and is comparable to the study of Evans and Carrie.[5]

The number of tops-ups required was significantly greater in the intermittent regimen. Fewer women required one or more top-ups in the continuous group (41%). This, however, is considerably more than in the studies of Davies and Fettes[8] (30%) and Bogod *et al.*[3] (24%) but this is probably because we included those top-up doses given for instrumental delivery. In addition, our top-ups were half the concentration of those given by Bogod *et al.*[3] This significant reduction in the need for a top-up in the continuous group, compared to the intermittent group, must reduce the risk of hypotension with subsequent CTG abnormality, both of which were less in the continuous regimen group. This may be why the Caesarean section rate was lower in the continuous group.

The neonatal outcome did not appear to be adversely affected by the continuous method and the number of babies who required admission to the special care baby unit was much less in the continuous group (Table 3).

It would appear that the use of a continuous infusion to maintain epidural analgesia during labour does not adversely affect obstetric or neonatal outcome and may

even have benefits over the traditional intermittent regimen. The continuous regimen is likely to be much less labour intensive due to the reduction in the number of top-ups required, the reduced incidence of hypotension and associated cardiotocographic evidence of intrapartum fetal hypoxia, and the rate of Caesarean section. Anaesthetists will continue to experiment to achieve an optimum concentration, infusion rate, total dose and type of local anaesthetic and this study encourages us to recommend a wider use of epidural analgesia by continuous infusion for pain relief in labour.

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Controlled-release morphine in cancer pain

Is a loading dose required when the formulation is changed?

P. J. HOSKIN, P. POULAIN AND G. W. HANKS

Summary

Nineteen patients with pain from advanced cancer stabilised on oral 4-hourly aqueous morphine who were to be converted to twice daily controlled-release morphine tablets (MST Continus) completed this study. Patients were randomised to receive either their usual 4-hourly morphine dose or placebo with the first dose of MST tablets. There were no significant differences between the treatment groups in ratings of pain intensity, pain relief or side effects, or in any of the measured pharmacokinetic parameters. No patients in the placebo group experienced any breakthrough pain, and nursing staff were unable to identify which patients had received the active dose of morphine elixir. We conclude that there is no need for a loading dose when aqueous morphine is changed to MST tablets.

Key words

Pain; chronic.
Analgesics, narcotic; morphine.

Controlled-release morphine tablets (MST Continus) provide a convenient way to deliver regular morphine in patients with pain from advanced cancer, and in a recent survey they emerged as the formulation of choice in this indication amongst both hospital doctors and general practitioners.[1] The controlled-release nature of this formulation makes it less appropriate for unstable pain and in patients who are first starting treatment with morphine, when oral aqueous morphine solution is preferred. This produces a peak serum concentration of morphine at 30–45 minutes compared with a delay of about 2.5 hours after administration of MST tablets.[2] It is therefore common clinical practice for patients to receive regular oral aqueous morphine 4-hourly until their pain is controlled and then to be converted to twice-daily controlled release tablets.

It has been demonstrated in a number of studies that oral solution and MST tablets are dose-equivalent,[2–4] so that the same 24-hour dose is given regardless of formulation. A potential problem exists, however, during the changeover period. There will be a lag period of 2–3 hours after the first dose of MST tablets before peak blood concentrations are achieved, and during which the effects of the previous dose of aqueous solution taken 4 hours before may be waning.

The purpose of this study is to determine whether this potential temporary decrease in blood concentrations has any clinical consequences and whether a final dose of aqueous morphine should be routinely administered with the first dose of MST tablets.

Method

Inpatients with advanced cancer who were receiving a regular 4-hourly aqueous oral morphine solution were entered into this study. Patients whose pain was controlled on a stable dose for at least the previous 5 days were eligible. The study was approved by the Ethics Committee of the Royal Marsden Hospital and consent was obtained from each patient after full explanation of the aims of the study and what would be involved. Exclusion criteria were a prognosis of less than 4 weeks, a total morphine dose greater than 800 mg daily, a serum haemoglobin of less than 9 g/dlitre, and inability to give informed consent.

Patients were prospectively randomised to receive either an additional dose of morphine solution or placebo with the first dose of MST tablets. Both preparations were prepared so as to have an identical taste and physical appearance, and the volume of each dose was standardised to 10 ml. The dose used for the active solution was the same as the regular 4-hourly dose. Randomisation was performed the day before the change to MST tablets. The active and placebo solutions were prepared freshly by the

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hospital pharmacy the day before administration and the randomisation code was kept in the hospital pharmacy so that the study remained double blind.

A forearm vein was cannulated and a baseline blood sample taken on the day of the study, followed by administration of the first dose of MST tablets, together with the additional dose of morphine solution or placebo. Venous blood was collected at half-hourly intervals for the first 7 hours, and then hourly for 5 hours until the next dose was due. Blood samples were collected into a glass tube and rolled for 30 minutes before centrifugation at 3000 rpm for 10 minutes. Serum was separated and stored at $-20°C$ until analysis.

During the 12-hour study period measurements were made of pain with a 100-mm visual analogue scale and a 4-point categorical scale, and of pain relief using a 100-mm visual analogue scale. Details of side effects were also collected using a 4-point categorical scale for nausea, vomiting, drowsiness, dry mouth, tremor, sweating and jerking. These scales were administered at 0, 4, 8, and 12 hours. The nurse who had looked after the patient for that day was asked to complete a short questionnaire at completion of the study day to indicate her own assessment of the patient's pain, side effects and whether the patient had received placebo or active drug with the first dose of MST tablets.

Analysis. Analysis of the serum samples for morphine was performed using a specific radioimmunoassay after liquid-solid extraction.[5] Goat antimorphine antiserum was obtained from Bioclin, Cardiff.

Statistical analysis. The area under the curve of serum concentration versus time (AUC) for the 12-hour study period was calculated using the trapezoidal method. Correlation between dose and AUC was calculated using the least squares method and comparison between the two treatment groups of pharmacokinetic and pharmacodynamic data was performed using Student's *t*-test.

Results

Twenty patients were randomised into the study. One patient was withdrawn on the day of the study; his condition had deteriorated during the previous 12 hours. Ten of the remaining 19 patients received an active dose of morphine solution with their first dose of MST tablets and nine received placebo. Details of the patients are shown in Table 1; these include their regular dose of morphine and the duration of treatment. The results for time to peak serum concentration (t_{max}), peak serum concentration (C_{max}), minimum serum concentration at time zero (C_{min}) and serum concentration at 1 hour (C_1) are shown in Table 2. In view of the wide range of doses used the effect of the loading dose on the serum concentration was explored by determining the ratios of C_{max}/C_{min}, C_1/C_{max} and C_1/C_{min} which are shown in Table 3.

Table 4 shows the actual values for $AUC_{(0\text{–}12)}$ in the two groups and values corrected to a standard dose of 100 mg morphine. A good correlation between AUC and dose was obtained ($r = 0.97$, $p < 0.001$ for placebo group and $r = 0.85$, $p < 0.001$ for the active group). Mean data for pain and pain relief scores are shown in Table 5. No statistically significant change in these scores over the 12-hour study period is seen and no significant change in side effect profile was seen (Table 6). Table 7 shows the results of the nursing assessment on completion of the study day.

Discussion

The population of patients entered into this study is representative of those with advanced cancer who require regular oral morphine for pain across a wide dose range. No significant effect was observed of a loading dose of morphine elixir given with the first dose of controlled-release morphine tablets. The patients' primary nurses were unable to identify which patients had received morphine

Table 1. Details of patients, duration of medication and morphine dose.

Patient	Age (years)	Primary tumour	Morphine dose (mg/24 hours)	Duration on morphine (days)	
				Overall	Current dose
Placebo					
1	65	Breast	60	169	169
2	62	Stomach	600	38	5
3	68	Anal canal	360	123	13
4	81	Breast	120	420	15
5	65	Breast	720	480	5
6	67	Prostate	120	72	14
7	73	Breast	250	150	10
8	72	Lung	60	11	11
9	75	Breast	42	9	6
Morphine elixir					
10	65	Colon	480	127	8
11	74	Bronchus	600	60	6
12	84	Penis	80	21	7
13	64	Unknown	100	9	9
14	74	Prostate	100	21	6
15	51	Lung	240	71	15
16	63	Lung	60	7	7
17	66	Ovary	120	42	5
18	72	Bronchus	180	150	9
19	65	Ovary	360	105	28

Table 2. Results for time to peak plasma concentration (t_{max}), peak serum concentration (C_{max}), minimum serum concentration (C_{min}) and serum concentration at 1 hour (C_1).

Patient	t_{max} (hours)	C_{max} ng/ml	C_{min} ng/ml	C_1 ng/ml
Placebo				
1	2.0	26.0	6.2	12.8
2	2.5	456.2	206.0	293.7
3	2.0	275.2	116.1	115.2
4	1.5	429.1	23.0	74.3
5	3.5	593.4	137.6	172.2
6	1.0	41.5	7.6	41.5
7	0.5	50.6	24.9	34.4
8	4.5	69.8	28.3	25.3
9	1.5	17.0	7.1	15.1
Median	2.0			
Morphine elixir				
10	2.0	203.4	32.9	95.2
11	0.5	511.6	174.5	489.4
12	1.5	48.8	9.6	42.5
13	0.5	50.6	24.9	34.4
14	1.0	60.1	18.3	60.1
15	3.5	198.0	55.5	161.8
16	5.5	34.0	5.8	8.9
17	1.0	134.5	24.5	134.5
18	3.5	56.4	29.5	33.7
19	0.5	348.5	64.1	251.1
Median	1.25			

elixir. Six out of eight patients who received placebo were thought by their nurses to have had active morphine solution and six out of 10 patients who received active solution were thought to have been given placebo. Both pain control and side effects were unchanged for most patients from the previous treatment during the changeover period.

Table 3. Ratios of C_{max}/C_{min}, C_1/C_{max} and C_1/C_{min}.

Patient	C_{max}/C_{min}	C_1/C_{max}	C_1/C_{min}
Placebo			
1	4.2	0.5	2.1
2	2.2	0.6	1.4
3	2.4	0.4	1.0
4	18.7	0.2	3.2
5	4.3	0.3	1.3
6	5.5	1.0	5.5
7	2.0	0.7	1.4
8	2.5	0.4	0.9
9	2.4	0.9	2.1
Mean	4.9	0.55	2.1
(SEM)	(1.8)	(0.09)	(0.5)
Morphine elixir			
10	6.2	0.5	2.9
11	2.9	1.0	2.8
12	5.1	0.9	4.4
13	2.0	0.7	1.4
14	3.3	1.0	3.3
15	3.6	0.8	2.9
16	5.9	0.3	1.5
17	5.5	1.0	5.5
18	1.9	0.6	1.1
19	5.4	0.7	3.9
Mean	4.2	0.75	3.0
(SEM)	(0.5)	(0.07)	(0.4)

Differences between placebo and active dose are not significant ($p > 0.05$).

Table 4. Absolute values for $AUC_{(0-12)}$ and values corrected to 100 mg administered dose.

Patient	$AUC_{(0-12)}$ ng/ml/hour	Corrected AUC ng/ml/hour
Placebo		
1	130.2	433.9
2	2837.6	945.9
3	1306.6	725.9
4	790.7	1317.8
5	2846.8	790.8
6	314.2	523.7
7	350.8	350.8
8	399.4	1331.5
9	125.8	629.0
Mean		783.2
(SEM)		(118.8)
Morphine elixir		
10	933.0	291.6
11	4129.6	1032.4
12	286.2	540.1
13	349.6	521.7
14	354.3	528.9
15	1591.4	994.6
16	148.3	370.7
17	542.0	677.5
18	459.4	382.9
19	1554.7	647.8
Mean		598.8
(SEM)		(78.9)

Difference between corrected AUCs not significant ($p > 0.05$).

The pharmacokinetic data do not show any significant differences between the treatment groups. The median t_{max} was slightly shorter with the loading dose of morphine (1.25 hours compared with 2.0 hours); and the ratio of C_1/C_{max} was slightly greater (0.75 compared with 0.55). The dose-adjusted AUCs were comparable.

The figures for the AUCs are high, particularly when compared with a recent study in healthy volunteers.[6]

Table 5. Mean pain and pain relief scores for placebo and morphine elixir groups during 12-hour study period.

	Time (hours) 0	4	8	12
Pain VAS score, mean (SEM)				
Placebo	14.3 (4.8)	18.7 (7.1)	18.8 (4.9)	19.1 (6.2)
Active	14.1 (5.7)	6.7 (2.4)	13.3 (4.3)	11.2 (3.8)
Pain relief VAS score, mean (SEM)				
Placebo	75.9 (7.5)	76.4 (7.2)	70.7 (8.7)	67.8 (8.7)
Active	86.8 (5.8)	83.6 (6.4)	79.4 (7.3)	86.4 (4.4)
**Pain category score, median (range)*				
Placebo	1 (0–2)	1 (0–3)	2 (0–3)	1 (0–3)
Active	0.5 (0–2)	0 (0–2)	0.5 (0–2)	0.5 (0–3)

Observed differences are not significant $p > 0.05$.
*4-point scale: no pain (0); mild (1); moderate (2); severe (3).

Table 6. Median scores (range) for side effects based on 4-point scale*.

		Time (hours)			
		0	4	8	12
Nausea	Placebo	0 (0–2)	0 (0–3)	0 (0–1)	0 (0–2)
	Active	0 (0–3)	0 (0–2)	0 (0–2)	0 (0–2)
Vomiting	Placebo	0 (0–1)	0 (0–3)	0 (0–1)	0
	Active	0 (0–3)	0 (0–1)	0 (0–2)	0 (0–2)
Drowsy	Placebo	0 (0–2)	1 (0–3)	1 (0–3)	0 (0–3)
	Active	1 (0–1)	1 (0–3)	1 (0–3)	1 (0–3)
Sweating	Placebo	0 (0–1)	0 (0–2)	0 (0–1)	0 (0–1)
	Active	0 (0–2)	0 (0–1)	0 (0–2)	0 (0–2)
Dry mouth	Placebo	1 (0–3)	0 (0–2)	1 (0–2)	0 (0–2)
	Active	1.5 (1–2)	1.5 (0–3)	1.5 (0–3)	1.5 (0–2)
Tremor	Placebo	0 (0–1)	0 (0–1)	0 (0–1)	0 (0–1)
	Active	1 (0–3)	1 (0–3)	0 (0–2)	1 (0–3)
Dizzy	Placebo	0 (0–1)	0 (0–1)	0 (0–2)	0 (0–2)
	Active	1 (1–2)	0 (0–1)	1 (0–1)	0 (0–1)

Differences within each group are not significant $p > 0.05$.
*0, none; 1, mild; 2, moderate; 3, severe.

Table 7. Nursing assessment of pain, side effects and whether placebo or active drug given.

	Placebo				Active		
	Pain	Side effects	Preparation		Pain	Side effects	Preparation
Patient				Patient			
1	—	—	—	10	2	2	P
2	2	2	A	11	2	2	P
3	1	3	A	12	2	3	A
4	2	2	A	13	2	3	A
5	2	2	A	14	2	2	A
6	2	2	P	15	2	2	A
7	2	3	P	16	2	2	P
8	2	3	A	17	2	2	A
9	1	2	A	18	2	2	P
				19	2	2	P

1, Better than previous 24 hours; 2, unchanged; 3, worse than previous 24 hours; P, placebo; A, active.

However, our patient sample was elderly (median 67 years, range 51 to 84 years) and at steady state. The volunteer study in comparison involved young morphine-naive subjects aged 21–40 years, and a single oral dose of morphine. We obtained figures similar to those in the present investigation in a recent study in elderly cancer patients[2].

The pain intensity VAS and the categorical scale both indicated lower scores in the active group particularly at 4 hours. However, none of these differences was statistically significant and the scores in both groups indicated very low levels of pain. None of the patients in the placebo group experienced any break-through pain particularly during the early part of the study day.

In conclusion, we have found no evidence that when changing from regular 4-hourly morphine elixir solution to regular 12-hourly controlled-release tablets, an additional dose of morphine solution is required to cover the theoretical lag period before significant amounts of morphine are absorbed from the controlled-release tablet.

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Intravenous regional analgesia with prilocaine for foot surgery

The effect of slow injections and high tourniquet inflation pressures

J. A. H. DAVIES

Summary

Intravenous regional analgesia for foot surgery with an ankle tourniquet was used for 48 cases. Prilocaine 0.5% 3 mg/kg body weight was injected either quickly over about 2 minutes or slowly over about 5 minutes. The tourniquet was inflated either to occlusion pressure plus 100 mmHg or to occlusion pressure plus 200 mmHg. Plasma prilocaine levels were measured while the tourniquet remained inflated and after release of the tourniquet. All four techniques resulted in a low incidence and magnitude of prilocaine leak and low prilocaine plasma levels after tourniquet release. The data suggest that a slow injection with the high tourniquet inflation pressure is better, although the differences in leakage with an intact tourniquet were not statistically significant. Excellent analgesia was achieved in over 90% of patients and there were no complete failures. No dangerously high prilocaine plasma levels were produced and no serious side effects observed.

Key words

Anaesthetic techniques; regional, intravenous.
Anaesthetics, local; prilocaine.

Intravenous regional analgesia (IVRA) was shown to be a satisfactory technique for foot surgery with tourniquets placed on the thigh,[1] just distal to the knee[2–4] or when placed just above the malleoli.[5] Bupivacaine and lignocaine were employed with success, but toxic effects[6–9] from these agents led to recommendations that they should no longer be used for IVRA.[6,10,11]

A leak of local anaesthetic past an inflated tourniquet during IVRA was demonstrated using bupivacaine,[12–14] and although the leak is usually well below that likely to produce toxicity,[5] under certain circumstances it can be large.[15] Prilocaine was recommended as the agent of choice[16,17] for IVRA and was used successfully for foot surgery,[2,18] but no reports have yet appeared which measured the leak into the systemic circulation while the tourniquet was inflated.

Fast injections that produce high venous pressure may be associated with an increased leak past the tourniquet,[19,20] whereas slow injections, in addition to their wisdom on general principles,[21] could be expected to result in less leak into the systemic circulation. Recommended tourniquet inflation pressures[22,23] vary widely and this is a sure indication that this aspect of the technique requires further study. It is possible that higher tourniquet inflation pressures would lead to a reduction in leakage.

To test this, IVRA for elective foot surgery was carried out using prilocaine 0.5%, and the leak measured using both fast and slow injections of prilocaine, and with the tourniquet inflated to two different pressures. Blood samples for prilocaine levels were also taken after release of the tourniquet at the end of the operation.

Method

Forty-eight patients who had IVRA with prilocaine 0.5% (3 mg/kg body weight) for elective foot surgery were allocated at random to four groups.

Fifteen of the operations were for correction of hallux valgus often combined with corrective operations on other toes; 14 were corrective operations for one or more toes. The remainder included operations for ganglions, metatarsal osteotomies, removal of exostoses and excision of toenails. Twenty-nine operations involved the left foot and 19 the right. Approval was given by the South Birmingham Health Authority Ethics Committee and informed consent was obtained from all patients before the procedure.

The groups received the following: Group 1: slow injection of prilocaine, and tourniquet inflation pressure of occlusion pressure plus 100 mmHg (15 cases). Group 2: fast injection of prilocaine and tourniquet inflation pressure of occlusion pressure plus 100 mmHg (15 cases). Group 3: slow injection of prilocaine and tourniquet inflation pressure of occlusion pressure plus 200 mmHg (nine cases). Group 4: fast injection of prilocaine and tourniquet inflation pressure of occlusion pressure plus 200 mmHg (nine cases).

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Ten of the 48 patients received either diazepam 10 mg or papaveretum 10 mg and hyoscine 0.2 mg. A 21-gauge butterfly indwelling needle was placed in a vein usually on the dorsum of the foot. Difficult veins were dilated by immersing the foot in warm water. An oval plastic mop bucket was used since it accommodates the foot better than a conventional bucket. A 2-mm indwelling cannula was placed in a vein in the antecubital fossa for sampling blood and for intravenous access.

A double cuff tourniquet (Biomet Ltd), each cuff width 6 cm, was applied just above the malleoli, over several turns of vellband, and the velcro fastening of the tourniquet was reinforced with several turns of elastoplast to provide extra security.

Occlusion pressure[15] was determined for each cuff by inflating the tourniquet until either the dorsalis pedis or posterior tibial arterial pulsation was no longer palpable. The limb was elevated for at least 2 minutes for exsanguination. The proximal tourniquet was inflated either to 200 or 100 mmHg above the highest determined occlusion pressure. The range of inflation pressures employed was between 240 and 600 mmHg. A bicycle type pump was used to inflate the tourniquet. A volume of prilocaine 0.5% plain without preservative (Astra) that corresponds to 3 mg/kg was then injected into the indwelling butterfly needle. A 16-gauge green needle was used for fast injections and a 25-gauge orange needle for slow. Use of a 60-ml syringe with a 25-gauge needle made the slow injection mandatory.

Eight-millilitre samples of venous blood from the antecubital fossa were taken at the end of the injection and then at 5, 10 and 15 minutes after the beginning of the injection. This gave four samples in the case of the fast injection, but only three for the slow injection, since the end of the injection was nearly 5 minutes after injection. The samples were put in lithium heparin containers, mixed well, centrifuged later that day, and the plasma frozen. They were later analysed for plasma prilocaine level by gas chromatography using a nitrogen detector sensitive to 0.1 μg/ml. The distal tourniquet was inflated to the same pressure as for the proximal tourniquet, and the proximal tourniquet was deflated, when needle prick demonstrated a good block. This was invariably 12 minutes after the start of injection and preceded the 15-minute sample by approximately 3 minutes.

The patient was transferred from the anaesthetic room to the operating theatre when the surgical team was ready and when good analgesia had developed. This was approximately half-an-hour after the start of the injection in the majority of cases. Surgical delays increased this in four cases to more than 40 minutes and in one case to 95 minutes. The operation started less than 25 minutes from start of injection in 13 cases. Patients could listen to music from headphones during the operation and most were pleased to do so. The tourniquet was deflated without re-inflation on completion of surgery and further blood samples obtained at 5, 10 and 15 minutes after deflation of the tourniquet.

Patients were asked their opinion about the procedure and whether they would like the technique repeated should they require a further operation. They were asked about symptoms or side effects, and observed in the theatre area for at least 15 minutes after the tourniquet was finally released. The surgeon was asked whether he found the technique satisfactory.

The efficacy of the technique was scored according to the following criteria. Score 1: good analgesia with no supplementary medication necessary; score 2: analgesia satisfactory but the patient was anxious and restless and required sedation with 5–10 mg diazepam; score 3: analgesia inadequate at some point during the operation and required supplementary local anaesthesia, usually 5–10 ml 1% lignocaine injected by the surgeon into areas that retained sensitivity; score 4: analgesia inadequate and inhalational anaesthesia required.

The groups were comparable with regard to sex (36 females and 12 males), age (average 52.2 years, range 20–81) and weight (average 61.8 kg, range 41–111 kg). Occlusion pressure for the proximal cuff ranged from 140 to 400 mmHg and for the distal cuff from 140 to 320 mmHg. The occlusion pressure for both cuffs in 37 cases was the same or differed by less than 20 mmHg. With greater differences the proximal cuff gave the higher value. In six cases this was 40 mmHg higher, in three 60 mmHg higher and in two 100 and 200 mmHg higher. These occasional large differences in occlusion pressure for the two cuffs underline the importance of measuring both.

The mean duration of the slow injection was 4 minutes and 53 seconds (range 4 minutes 11 seconds–6 minutes 24 seconds) equivalent to a mean injection speed of 0.12 ml/second (range 0.09–0.16 ml/second). The mean duration of the fast injection was 1 minute and 4 seconds (range 1 minute 15 seconds–2 minutes 13 seconds) equivalent to a mean injection speed of 0.39 ml/second (range 0.126–0.51 ml/second).

The time from start of injection to final tourniquet release varied in the four groups. Group 1: mean time 55 (range 34–80) minutes; Group 2: mean time 47 (range 26–117) minutes; Group 3: mean time 38 (range 37–60) minutes; Group 4: mean time 66 (range 36–123) minutes.

Results

Leakage of prilocaine past the inflated tourniquet (see Tables 1 and 2) occurred in 14 cases (29%) and was not detected in 34 cases. In samples where a leak was present the highest plasma prilocaine level was 0.95 μg/ml with a mean level of 0.28 μg/ml (range 0.1–0.95 μg/ml). The proportion of cases in which a leak occurred and the number of samples that contained prilocaine was greater in Group 2, but did not reach statistical significance (Fisher's exact test). The amount of the leak was not the same in the four groups. Fast injection at both tourniquet inflation pressures associated with a higher mean and peak plasma prilocaine level was not statistically significantly (Unpaired *t*-test) different from a slow injection. Prilocaine was present in the plasma in all cases after tourniquet release, and in all except five samples (see Tables 3 and 4). The mean plasma level was 0.37 μg/ml with a range of 0.1–1.3 μg/ml. Group 3 with a slow injection and tourniquet inflation pressure of occlusion +200 mmHg gave the lowest mean and peak plasma prilocaine level (mean 0.21 μg, range 0.11 μg–0.36 μg/ml). Group 3 plasma levels were significantly less than Group 1 ($p < 0.01$) and less than Group 2 ($p < 0.05$). Group 3 plasma levels were not statistically significantly less than Group 4.

Good analgesia (see Table 5) was obtained in 44 of the 48 cases (92%) (score 1). In only one case did anxiety and restlessness require supplemental sedation (score 2). In

Table 1. Prilocaine leak past inflated tourniquet.

Group		Number of cases	Cases with leak	% cases with leak	Mean and range* plasma level μg/ml
1	Slow injection occlusion +100 mmHg	15	4	27	0.23 (0.18–0.36)
2	Fast injection occlusion +100 mmHg	15	6	40	0.36 (0.1–0.95)
3	Slow injection occlusion +200 mmHg	9	2	22	0.205 (0.13–0.28)
4	Fast injection occlusion +200 mmHg	9	7	22	0.34 (0.11–0.84)
	All cases	48	14	29	0.28 (0.10–0.95)

* Excluding samples with no leak.

Table 2. Proportion of samples that showed a leak with the tourniquet inflated.

Group	Number of samples	Samples that contained prilocaine
1	45	11 (24.4%)
2	60	20 (33.3%)
3	27	4 (14.8%)
4	36	6 (16.7%)

three cases additional local anaesthetic was infiltrated into the wound (score 3). None required general anaesthesia (score 4).

Discussion

A leak of local anaesthetic agent with IVRA for foot surgery after injection while the tourniquet remained inflated was demonstrated previously;[13] in a series by the author that employed bupivacaine 0.25% it occurred in all cases.[5] A leak was present in 14 cases out of 48 (29%) in the present series, which implies that even with a careful technique with the higher cuff inflation, pressure of occlusion pressure plus 200 mmHg and with a slow injection, leakage can be expected in a proportion of cases. The mean magnitude of prilocaine leak overall was 0.28 μg/ml with a range of 0.1–0.84 μg/ml if samples with no leak are excluded. This low plasma prilocaine level is well below that associated with toxicity which is probably about 6 μg/ml.[24] Such low levels give no cause for anxiety.

The incidence and magnitude of leaks was not uniform in the four groups. The greatest incidence of leak, 40% of cases and 33% of blood samples, occurred in Group 3 with the fast injection and the low tourniquet inflation pressure; the lowest incidence occurred in Groups 3 and 4 with the higher tourniquet inflation pressure. The data also suggest that the greatest magnitude of leak occurred in Groups 2 and 4 with the fast injection at both tourniquet inflation pressures, and that the lowest leak occurred in Groups 1 and 3 with the slow injection at both tourniquet inflation pressures.

The data suggest that tourniquet inflation pressure determines the incidence of leakage, and the speed of injection its magnitude, but none of these differences reached statistical significance in this study and no definitive conclusions can be drawn. It is estimated that some 400 cases are required to test for significance, on the data obtained in this study; only a large multicentre trial could provide such numbers. The data considered together may suggest that the combination of a fast injection with a low tourniquet inflation pressure (Group 2) is likely to produce the greatest leak and is better avoided. They also suggest that the slow injection with a high tourniquet inflation pressure (Group 3) produces the lowest leak and is the preferred technique.

A large syringe (60 ml) and a fine needle (25 gauge) will ensure that the injection is slow and prevents inadvertent fast injection. The mean prilocaine plasma level after release of the tourniquet at the end of the operation was 0.37 μg/ml with a range of 0.1–1.3 μg/ml. This was higher than leakage levels recorded with the tourniquet inflated, but these plasma prilocaine levels are still well below those associated with toxicity.[24] The reason for the low level in

Table 3. Prilocaine plasma levels after tourniquet release.

Group		Number of cases	Plasma level μg/ml mean and range	95% confidence interval
1	Slow injection occlusion +100 mmHg	15	0.446 (0.14–1.3)	0.325–0.557
2	Fast injection occlusion +100 mmHg	15	0.424 (0.11–1.02)	0.302–0.546
3	Slow injection occlusion +200 mmHg	9	0.198 (0.11–0.36)	0.127–0.269
4	Fast injection occlusion +200 mmHg	9	0.353 (0.1–0.94)	0.160–0.546

Table 4. Prilocaine plasma levels after tourniquet release—statistical evaluation.

Mean plasma level	Difference between means (SEM)	DF	Level of significance
Group 1 > Group 3	0.249 (0.0749)	19	1%
Group 2 > Group 3	0.226 (0.0815)	21	5%
Group 4 > Group 3	0.155 (0.0932)	15	N.S.

Group 3 is not clear but may reflect a smaller leak which has occurred previously during the injection with the tourniquet inflated. The plasma prilocaine levels in Group 3 were significantly lower than in Groups 1 and 2. These data provide evidence that Group 3 with a high tourniquet inflation pressure and a slow injection will lead to the lowest plasma prilocaine levels when the tourniquet is released.

Good analgesia (score 1) was present in 44 of the 48 cases (92%) and was excellent but for a few cases who complained of minor discomfort, usually associated with the cuff, who responded well after reassurance. One case (score 2) showed signs of anxiety and restlessness but settled after receiving intravenous diazepam 7.5 mg. One of the three cases who required supplemental infiltration of local anaesthesia (score 3) felt discomfort during skin incision but did not withdraw her foot. Her wound was infiltrated with 15 ml 0.5% lignocaine during the course of the operation and she complained several times of feeling discomfort but no pain. She said she was pleased with the technique, at the end of the operation, and would like the same technique for a future operation. Another two cases were given 1% lignocaine 5 ml when they complained of soreness. Both were also quite happy to have the same technique again.

Thirty-five of the 48 cases expressed a clear preference for the same technique to be repeated if a further operation proved necessary. One patient wanted to see the operation performed! He was happy that the technique allowed him to do this.

The surgeons were pleased with the technique although there is no doubt that occasionally venous oozing occurred which did not present a serious surgical problem. Better exsanguination might give a better bloodless field. The success rate achieved is better than with most other local anaesthetic techniques and the plasma prilocaine levels are much less than those demonstrated with other local anaesthetic techniques.[25,26] Systemic toxicity from the agent is unlikely to occur if careful attention to the details of the technique are observed. All of the four methods tested gave good clinical analgesia and acceptably low prilocaine plasma levels, and it is suggested that even in the absence of statistical confirmation a slow injection of prilocaine with a tourniquet inflation pressure of occlusion pressure plus 200 mmHg may be the preferred technique.

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Table 5. Success with technique.

Group		Number of cases	Score 1	Score 2	Score 3	Score 4
1	Slow injection occlusion + 100 mmHg	15	14	0	1	0
2	Fast injection occlusion + 100 mmHG	15	14	0	1	0
3	Slow injection occlusion + 200 mmHg	9	7	1	1	0
4	Fast injection occlusion + 200 mmHg	9	9	0	0	0

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Cardiovascular effects of fibrescope-guided nasotracheal intubation

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Summary

The cardiovascular effects of fibrescope-guided nasotracheal intubation were compared to those of a control group of patients who were intubated using the Macintosh laryngoscope. The 60 patients studied received a standard anaesthetic technique which included a muscle relaxant and were allocated randomly to one of two groups immediately before tracheal intubation. Systolic and diastolic arterial pressures in the fibreoptic group were significantly lower than in the control group during the first minute after intubation. The maximum increase in diastolic pressure was significantly lower in the fibreoptic group. The heart rate in the fibreoptic group was significantly higher than in the control group during all five minutes after intubation. The maximum increase in heart rate was significantly higher in the fibreoptic group. The cardiovascular responses to fibreoptic nasotracheal intubation under general anaesthesia should not cause undue concern in fit patients, but appropriate measures should be taken to prevent excessive tachycardia in compromised patients.

Key words

Anaesthesia; general.
Intubation, tracheal; techniques.

Stimulation of the upper respiratory tract by laryngoscopy and tracheal intubation induces sympathetically-mediated tachycardia and hypertension.[1,2] This pressor response can cause significant morbidity and mortality, and may be hazardous particularly for patients with cerebral or cardiovascular disease.[3,4] It has been reported recently that fibreoptic orotracheal intubation under general anaesthesia produces a more severe pressor response than conventional intubation using the Macintosh laryngoscope.[5]

Nasotracheal rather than orotracheal intubation is indicated in anaesthesia for intra-oral operations, when the presence of a transoral tube may obstruct surgical access. The cardiovascular changes appear to be minimal when fibreoptic nasal intubation is performed under local anaesthesia in awake but sedated patients.[6] However, the responses to fibreoptic nasal intubation under general anaesthesia have not been investigated.

There is evidence that the magnitude of the pressor response varies with the intubation technique;[5–7] consequently, it is important to establish the cardiovascular effects of fibreoptic-assisted nasal intubation in anaesthetised patients. This study was designed to identify the heart rate and arterial pressure changes during nasal intubation effected with the fibreoptic laryngoscope and to compare them with those of nasal intubation effected with the Macintosh laryngoscope during general anaesthesia.

Patients and methods

The investigation was approved by the ethics committee of the South Birmingham Health Authority and informed written consent was obtained from each patient. Sixty fit (ASA class 1) patients aged between 16 and 48 years who required elective nasotracheal intubation for oral surgery under general anaesthesia were studied. Patients taking vasoactive drugs and those expected to present difficulty at intubation were excluded from the trial. The majority were scheduled for either the excision of impacted third molar teeth or the enucleation of maxillary cysts.

Patients were premedicated orally with temazepam 20 mg, one hour before the operation. An intravenous cannula was sited and continuous ECG monitoring established in the anaesthetic room. Arterial pressure and heart rate were recorded at one-minute intervals with a Dinamap 1846 monitor and TR2000 printer. A 5-minute stabilisation period was allowed before baseline measurements were made. Anaesthesia was induced with a dose of thiopentone sufficient to obtund the eyelash reflex (usually 4–5 mg/kg over 30 seconds); then vecuronium was administered in a dose of 0.125 mg/kg. The patient's lungs were ventilated for 4 minutes with oxygen 50%, nitrous oxide 50% and isoflurane 1% by means of a Guedel airway and a facemask attached to a Bain system with an initial fresh gas flow rate

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Table 1. Age, sex and weight characteristics. Data expressed as mean (SEM) where applicable.

	Age (years)	Weight (kg)	Sex
Control group ($n=30$)	24.2 (1.0)	65.7 (2.2)	10M 20F
Fibreoptic group ($n=30$)	25.5 (1.3)	65.6 (2.3)	10M 20F

Table 2. Time to achieve successful intubation.

Group	Number	Mean (SEM) time, seconds	Range, seconds
Control	29	30 (1.6)	19–58
Fibreoptic	30	37 (1.6)*	25–57

*$p<0.05$ compared to control group.

of 90 ml/kg minute. A Datex Normocap capnometer sampling tube was placed in the airway and fresh gas flow rate and ventilation were adjusted to maintain the end-expired carbon dioxide concentration at 5–5.5%.

Immediately before nasotracheal intubation, patients were allocated, using random numbers, to either the control group, who were intubated using a Macintosh laryngoscope, or to the fibreoptic group, who were intubated using an Olympus LF1 fibreoptic laryngoscope. In each case, a well-lubricated Leyland red-rubber cuffed nasal tube ('Cuftrac') was used (internal diameter 7.5 mm for males and 6.5 mm for females).

The tube was placed into the trachea under direct vision in the conventional manner using a Macintosh laryngoscope[8] with the aid of Magill's forceps if necessary in the control group. Fibreoptic intubation was undertaken in a manner similar to that described by Witton,[9] except that a clear airway was maintained by a trained assistant who extended the atlanto-occipital joint and displaced the mandible anteriorly by application of firm pressure behind the ascending rami. (The protocol also allowed for direct anterior traction on the tongue, at the discretion of the operator, if airway clearance was otherwise inadequate, but it was not necessary to exercise this option in any patient.) Care was taken to ensure that the tip of the fibrescope advanced no more than 7.5 cm below the glottis, in order to avoid stimulation of the carina. The tracheal tube was rotated anticlockwise through approximately 90° as the bevel negotiated the larynx in order to avoid impaction on the arytenoids or the vocal cords, as has been described previously.[5] All the fibreoptic intubations were performed by one author (J.E.S.) who was experienced in the technique; the conventional intubations were undertaken in approximately equal proportions by all four authors.

The time to successful intubation was taken as the interval between removal of the facemask from the patient's face and reconnexion of the Bain system to the catheter mount after the intubation had been completed. The patient was withdrawn from the trial and the cardiovascular data obtained were not included in the results if any intubation could not be completed within 60 seconds.

Data were analysed by the paired Student's t-test (within groups) and the unpaired Student's t-test (between groups); $p < 0.05$ was considered significant.

Results

The two groups were similar in respect of age, weight and sex (Table 1).

Details of the times to successful intubation are shown in Table 2 and a frequency histogram is shown in Figure 1. Twenty-nine of the 30 conventional intubations were completed within one minute but visualisation of the larynx was difficult in one male patient, and a second attempt was necessary before intubation was achieved. All the fibreoptic intubations were completed within one minute, but the mean intubation time (37 seconds) was significantly greater than that of the control group (30 seconds).

Mean systolic arterial pressures before induction and before intubation were similar in each group (Table 3). Tracheal intubation caused significant increases in mean systolic pressure compared with pre-induction values in each group. Mean systolic pressure remained significantly elevated for 4 minutes after intubation in the control group and for all 5 minutes after intubation in the fibreoptic group. The highest mean systolic pressure was recorded during the first minute after intubation in the control group and the mean systolic pressure in the fibreoptic group was then significantly lower than that in the control group. The highest mean systolic pressure in the fibreoptic group was delayed until the second minute, when, and after which, there were no significant differences between the two groups. The mean diastolic arterial pressures followed a similar pattern.

Mean heart rates before induction and before intubation were similar in each group (Table 4). There were significant increases in mean heart rate after induction of anaesthesia in both groups compared to pre-induction values. Tracheal

Table 3. Mean (SEM) values of systolic and diastolic arterial pressures (mmHg) in control and fibreoptic groups.

Group	Before induction	Before intubation	Time after intubation, minutes				
			1	2	3	4	5
Control ($n=29$)							
Systolic	124 (2)	120 (3)	156 (4)†	150 (4)†	139 (3)†	130 (3)†	124 (3)
Diastolic	67 (1)	65 (2)	102 (2)†	88 (3)†	78 (2)†	72 (2)†	68 (1)
Fibreoptic ($n=30$)							
Systolic	120 (2)	118 (3)	146 (3)†*	153 (3)†	138 (3)†	131 (3)†	127 (3)†
Diastolic	67 (1)	69 (2)	96 (2)†*	90 (2)†	79 (2)†	75 (2)†	71 (1)†

†$p<0.05$ compared to pre-induction values; *$p<0.05$ compared to control group.

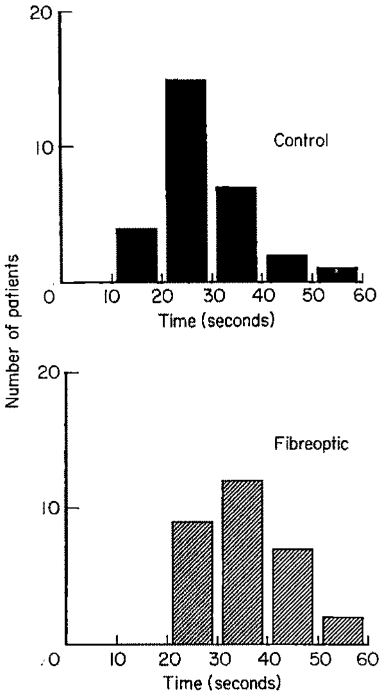

Fig. 1. Frequency histograms of times to achieve successful nasotracheal intubation in the control and fibreoptic groups.

intubation caused further significant increases in mean heart rate in both groups compared with pre-intubation values. The increase in heart rate compared to pre-intubation values was sustained for 4 minutes in the control group and for 5 minutes in the fibreoptic group. The mean heart rate in the fibreoptic group was significantly greater than in the control group during each of the 5 minutes after intubation.

The mean maximum increase in diastolic pressure from pre-intubation value in the fibreoptic group was 30 mmHg; this was significantly smaller than the corresponding increase in the control group (37 mmHg; Table 5). The mean maximum increase in heart rate from pre-intubation value in the fibreoptic group was 23 beats/minute, which was significantly greater than that in the control group (16 beats/minute).

Discussion

There is a consensus of opinion that the safest way to manage a known or suspected difficult intubation is to secure the airway using local anaesthesia with the patient awake.[10-12] Administration of general anaesthesia may result in upper airway obstruction, which may not be readily correctable by simple measures such as manipulation of the mandible or insertion of a pharyngeal airway.

Table 5. Mean (SEM) maximum increases in systolic and diastolic pressures (mmHg) and heart rate (beats/minute) from pre-intubation values.

Group	Systolic	Diastolic	Heart rate
Control ($n=29$)	42 (3)	37 (2)	16 (2)
Fibreoptic ($n=30$)	39 (2)	30 (2)*	23 (2)*

*$p<0.05$ compared to control group.

This situation is hazardous particularly for patients in whom tracheal intubation cannot be effected rapidly. Consequently, oxygenation is best assured in conscious patients, breathing spontaneously, who can maintain the patency of their own airway.

Fibreoptic endoscopy, in experienced hands, has emerged over recent years as an effective technique for awake intubation. It is likely to be more successful than rigid laryngoscopy and also more acceptable to the patient.[7,13,14] However, in the early stages, the failure rate with the instrument is high and considerable expertise must be acquired before anaesthetists can expect to achieve success in difficult cases. Equipment,[15-17] techniques[9,18] and training programmes[19] have been devised to help clinicians develop the necessary skills in a systematic way. One authority[19] emphasises the teaching of fibreoptic laryngoscopy on awake patients, although others advocate training on routine cases under general anaesthesia. Consequently, it is possible that an increasing number of fibrescope-guided intubations will be carried out under general anaesthesia as fibreoptic apparatus becomes more widely available and more anaesthetists recognise its value. It is well documented that the haemodynamic disturbances produced by conventional tracheal intubation can be harmful; thus it is particularly important to investigate the cardiovascular effects of this relatively new technique.

This study has shown that the hypertensive response to fibreoptic nasotracheal intubation under general anaesthesia is less severe than that associated with the conventional Macintosh technique. However, the fibrescope produces a significantly greater and more sustained tachycardia. These differences are probably of little consequence in healthy patients, but control of heart rate can be a critical factor in the anaesthetic management of patients with cardiovascular disease and therefore the technique should be avoided in patients compromised in this way. Alternatively, should fibreoptic endoscopy prove necessary to facilitate tracheal intubation during general anaesthesia in such patients, it is recommended that the pressor response be attenuated by pharmacological means.

The findings reported here contrast with those of a

Table 4. Mean (SEM) values of heart rate (beats/minute) in control and fibreoptic groups.

Group	Before induction	Before intubation	Time after intubation, minutes 1	2	3	4	5
Control ($n=29$)	76 (2)	84 (2)	97 (2)†	96 (2)†	94 (2)†	89 (2)†	85 (2)
Fibreoptic ($n=30$)	77 (3)	88 (2)	105 (3)†*	104 (3)†*	103 (3)†*	100 (3)†*	97 (3)†*

†$p<0.05$ compared to pre-intubation values; *$p<0.05$ compared to control group.

previous study which examined the haemodynamic responses to fibreoptic oral intubation under general anaesthesia.[5] In that study, both arterial pressure and heart rate in the fibreoptic group were greater than in control patients, whereas in the present study the arterial pressure was lower in patients intubated using the fibreoptic laryngoscope. These differences may arise because of the combined effects of differences in airway stimulation and differences in the duration of laryngoscopy between the two techniques. The fibrescope may produce less mechanical pressure on the tissues of the anterior pharynx, which may therefore induce less reflex sympathetic activity. However, fibreoptic laryngoscopy takes longer to accomplish than Macintosh laryngoscopy and this may tend to produce more sympathetic activity; it has been demonstrated that increasing the duration of laryngoscopy causes an increased hypertensive response.[20] During orotracheal intubation, the fibreoptic intubation time was approximately three times greater than that required using conventional laryngoscopy; thus, any benefit from reduced pharyngeal stimulation was outweighed by the effects of prolonged intubation. During nasotracheal intubation, the fibreoptic intubation time was only 25% greater than that required using the conventional technique. Decreased pharyngeal stimulation may have been a more important factor, resulting in partial attenuation of the hypertensive response. The marked tachycardia which occurred in both oral and nasal fibreoptic groups compared with controls may indicate that the heart rate response is more sensitive than the arterial pressure response to the effects of prolonged or fibreoptic intubation.

This trial has demonstrated the haemodynamic changes associated with fibreoptic nasotracheal intubation under general anaesthesia. Fibreoptic endoscopy is an important advance in airway management and is being adopted by an increasing number of anaesthetists. The pressor responses to the technique should not cause concern in healthy patients, but appropriate measures should be taken to avoid the tendency to tachycardia in susceptible patients.

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Anaesthesia, 1989, Volume 44, pages 911–912

CASE REPORT

Hyperosmolality due to antacid treatment

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Summary

Magnesium trisilicate mixture is an antacid used commonly in our Intensive Care Unit in the prevention and treatment of stress ulcers. In this case the administration of large doses over a period of time led to the development of massive hyperosmolality, cerebral dehydration and coma. Management with hypotonic fluid resulted in complete recovery.

Key words

Gastrointestinal tract; antacids.
Blood; osmolality.

Case history

A 60-year-old male was involved in a road traffic accident and sustained severe injuries to the cervical vertebrae and thoracic cage. He developed a pneumothorax that resulted in cardiac arrest which was treated successfully. Subsequently quadriplegia was noted and long term ventilatory support was required via a tracheostomy. Apart from a short episode of jaundice of uncertain aetiology, his general condition showed improvement with time and all vital observations, including his conscious level, were stable.

He received enteral nutrition through a nasogastric tube, and 2 months after the accident he had severe melaena and haematemesis, although he was on cimetidine 400 mg twice daily. His haemoglobin concentration was found to be 46 g/litre; gastroscopy revealed stress ulceration. Emergency truncal vagotomy and gastroduodenostomy was performed. His dose of cimetidine was increased after operation to 400 mg four times daily. He had another severe melaena and haematemesis on the fifth postoperative day, and the surgeon prescribed 100 ml/hour of magnesium trisilicate mixture (MTS) in an attempt to stop the bleeding.

The patient was found unconscious 22 hours later. Examination of his treatment regimen showed that a total of 2200 ml of MTS mixture had been given. Laboratory investigations revealed hyperosmolality (400 mosmol/kg) (Table 1).

MTS mixture contains 6 mmol of sodium per 10 ml according to the *British National Formulary*. This patient had therefore received, in the course of 22 hours, 1320 mmol of sodium. It was considered that the gross hyperosmolality was due to the MTS mixture and corrective treatment was started. This consisted of intravenous 0.45% NaCl in glucose 5%, in a volume calculated on the basis of body weight and serum sodium according to the equation:[1]

volume (in litres) to be replaced =

$$\mathrm{TBW}\frac{\text{serum sodium} - 140}{140},$$

Where TBW = total body water.

The total fluid volume infused over 72 hours included daily requirements and was guided by monitoring of serum electrolyte, urea and creatinine concentrations, blood glucose concentration, serum osmolality and assessment of

Table 1. Changes in serum electrolyte, urea and creatinine concentrations, blood glucose concentration, serum osmolality and urine output before, during and after administration of MTS mixture. The MTS mixture was administered during days 5 and 6.

Days	1	2	3	4	5	6	7	8	9
Sodium (mmol/litre)	144.0	152.0	141.0	135.0	168.0	171.0	158.0	148.0	137.0
Potassium (mmol/litre)	5.8	5.0	3.7	3.8	4.2	3.3	4.2	3.6	3.5
Urea (mmol/litre)	9.1	11.8	7.0	8.2	39.0	57.0	45.0	29.0	12.0
Glucose (mmol/litre)	6.0	4.9	12.0	9.0	8.0	12.0	7.8	8.6	7.0
Creatinine (μmol/litre	150.0	120.0	160.0	110.0	230.0	300.0	270.0	160.0	130.0
Osmolality (mosmol/kg)	299.0	316.0	295.0	290.0	370.0	400.0	358.0	325.0	285.0
Urine output (litres/day)	1.7	2.0	1.8	1.6	3.5	0.5	4.5	5.0	4.7

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his conscious level. The patient's general condition had improved by the end of the third day and his conscious level returned to normal. All laboratory investigations also returned to normal values.

Discussion

The causes of pure-solute-gain hypernatraemic hyperosmolality are usually accidental e.g. salt poisoning in infants, or iatrogenic e.g. high doses of sodium bicarbonate at cardiac arrest. The sodium load from MTS mixture in this case is an iatrogenic cause, the immediate consequences of which led to contraction of the intracellular compartment and cerebral dehydration. Iso-osmolar fluid in the form of glucose 5% or even sodium chloride 0.9% can be infused to correct the hypertonic state. This fluid should be given gradually as rapid correction may result in cerebral oedema.[2]

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Anaesthesia, 1989, Volume 44, pages 913–915

CASE REPORT

Chlormethiazole sedation for critically ill patients in renal failure

P. A. GRAY AND G. R. PARK

Summary

Chlormethiazole infusions were used successfully to provide night sedation for 10–19 nights in three patients with renal failure managed by continuous veno-venous haemofiltration with dialysis. Fluid overload has accompanied the use of this drug previously because of its low concentration. The ability to remove large amounts of fluid during haemofiltration dialysis proved to be effective in preventing this. All three patients had impaired liver function and showed evidence of chlormethiazole accumulation after 4–6 days. The combination of progressive reduction in dose and daily withdrawal of infusions prevented a major problem. Acceptance of this technique by the patients was high. Chlormethiazole may be a useful addition to the drugs available to provide sedation in well defined clinical circumstances.

Key words

Hypnotics; chlormethiazole.
Kidney; failure.

Infusions of chlormethiazole are well established in the management of alcohol withdrawal, status epilepticus, pre-eclampsia and short term sedation. The drug has been used also for prolonged infusion in critically ill patients,[1] in whom it can produce effective sedation and permit artificial ventilation whilst preserving cardiovascular stability. Modig[2] has suggested that chlormethiazole may have a protective effect in endotoxic shock, minimising cardiovascular and pulmonary instability, which could be advantageous in the critically ill patient.

Intravenous administration of chlormethiazole by bolus doses or by short infusions produces effective sedation with rapid onset and recovery due to redistribution into a large volume. It is eliminated mainly by hepatic metabolism, with less than 1% of unchanged drug excreted in the urine.[3] Saturation of redistribution sites may occur during long term infusions; its effect then is terminated by metabolism alone.[4] This problem may be exacerbated in the elderly,[5] the critically ill[1] and in patients whose hepatic function is chronically impaired.[6] Scott *et al.*[1] have suggested that the decrease in metabolism may be due to a reduction in hepatic blood flow. Thus recovery from long term infusions (more than 24–48 hours) may be prolonged significantly in some patients.

Chlormethiazole is formulated for intravenous use as a 0.8% solution in 4% glucose buffered with approximately 30 mmol sodium hydroxide to achieve a pH in the normal range. It is not available in a more concentrated form because the incidence of venous thrombophlebitis is unacceptably high and it may produce haemolysis if concentrations of 5% or more are used.[7] Thus, a large fluid load may be required if chlormethiazole is used for prolonged sedation. This feature has limited its usefulness, particularly in patients with renal failure and those with fluid and electrolyte problems.

Haemofiltration[8] removes fluid and small molecular weight substances (approximate molecular weight < 20 000) and can remove large volumes of fluid. The patient's own blood pressure can be used to drive the blood around the circuit (continuous arteriovenous haemofiltration; CAVH) if arterial and venous lines are inserted. Alternatively, two venous lines with a blood pump can be used to circulate the blood (continuous veno-venous haemofiltration; CVVH). Large volumes of haemofiltrate may be removed and replaced with a suitable solution. Continuous arteriovenous haemofiltration with dialysis (CAVHD) combines low volume arteriovenous haemofiltration with continuous perfusion of the filter using a haemodialysis solution.[9] Continuous veno-venous haemofiltration with dialysis (CVVHD), like CVVH, uses two venous lines and a blood pump, and provides a high and consistent flow through the membrane. These latter two techniques allow slow, gentle, continuous haemodialysis as well as control of fluid balance.

We describe three patients in whom CVVHD allowed the use of chlormethiazole.

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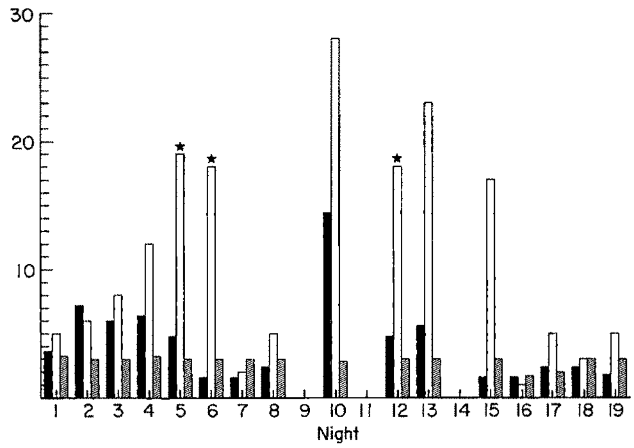

Fig. 1. Drug dose (mg), ■; recovery time (hours), □; sedation score, ▨; for 19 nightly chlormethiazole infusions in patient 1. *Marks the three occasions when patient 1 had not recovered noticeably before the infusion was inadvertently recommenced.

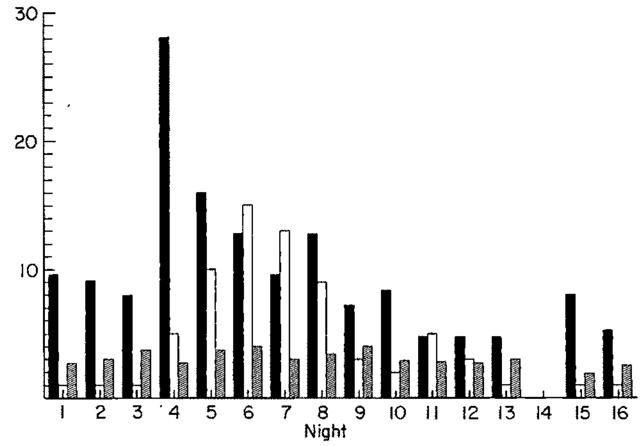

Fig. 2. Drug dose (mg), ■; recovery time (hours), □; sedation score, ▨; for 16 nightly chlormethiazole infusions in patient 2.

Case histories

The first patient, a 49-year-old male, had undergone orthotopic liver transplantation for cirrhosis due to α_1-antitrypsin deficiency. This had been complicated by the development of acute lung injury and acute renal failure 12 days after operation. The second patient, a 62-year-old male, had undergone resection of an abdominal aortic aneurysm. Subsequently, the left femoral graft and mesenteric artery became occluded and this caused infarction of the large bowel. Acute renal failure developed 2 days after operation. The third patient, a 44-year-old female, had undergone orthotopic liver transplantation for primary biliary cirrhosis. The patient had deteriorating renal function before operation, due to the hepatorenal syndrome, and developed acute renal failure on the second day after operation. All three patients required continuous mandatory ventilation (CMV) or synchronised intermittent mandatory ventilation (SIMV) and CVVHD. Abnormal liver function, predominantly obstructive in nature, developed in patients 1 and 2. Patient 3 had abnormal liver function (primarily hepatocellular in nature) which improved significantly during the period of chlormethiazole infusions (see Table 1).

Each night, chlormethiazole infusions were given to the patients to facilitate sleep. Patient 1 received the drug from the 21st to the 39th day, patient 2 from the 13th to the 29th day and patient 3 from the 10th to the 19th day in the Intensive Care Unit. Previously, sedation had been with bolus doses of morphine and midazolam given when required, but this regimen proved difficult to control. Propofol infusions were not used because we have observed that this method of sedation in deeply jaundiced patients may result in bradyarrhythmias (unpublished observation). The rate of the chlormethiazole infusions were controlled by the nursing staff within prescribed dose limits. The infusions were stopped during the day to encourage a normal circadian rhythm and to prevent drug accumulation. Sedation was assessed by the nursing staff each hour using the sedation score routinely employed in the intensive care unit:[10] 1, fully alert; 2, roused by voice; 3, asleep; 4, roused by pain; 5, unrousable. This simple scoring system may lack the sensitivity of more complex systems but has proved effective and consistent in clinical use.

The following information was recorded for each infusion: the infusion rate; the total dose of chlormethiazole given; the average sedation score during the infusion; and the time taken for the patient to reach maximum awakening after termination of the infusion. Figures 1–3 show these data for the three patients.

Adequate night sedation was defined as a score of 2 or 3. This objective was not achieved on only two occasions in the total of 45 nightly infusions. However, excessive sedation (score more than 3) occurred on six occasions, and on three occasions patient 1 had not recovered noticeably before the infusion was inadvertently restarted (marked with * in Fig. 1). It was necessary to withhold sedation for patient 1 on three occasions, and on one occasion for patient 2, because of oversedation.

Recovery time increased rapidly after sedation on the first 6 nights in all patients and improved only when the drug dose was reduced (usually achieved by a decrease in the duration of the infusion to approximately 3 to 4 hours rather than a change in the rate of infusion). Recovery

Table 1. Range of liver function tests during the period of chlormethiazole infusion.

	Patients 1	Patients 2	Patients 3	Normal range
Bilirubin (μmol/litre)	240–466	15–55	45–97	2–17
Alkaline phosphatase (units/litre)	88–460	108–322	158–224	30–135
Alanine aminotransferase (units/litre)	28–51	19–48	33–203	7–40

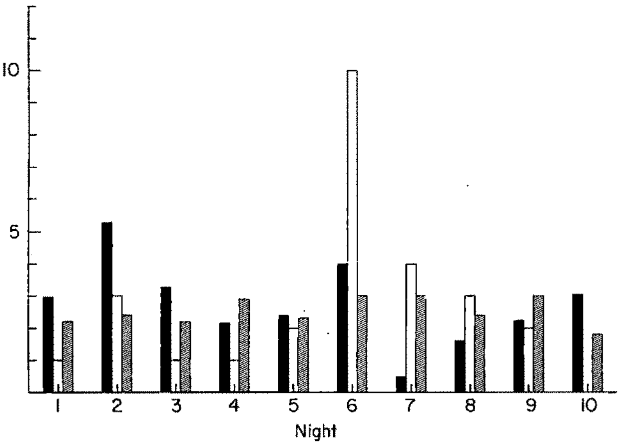

Fig. 3. Drug dose (mg), ■; recovery time (hours), □; sedation score, ▨; for 10 nightly chlormethiazole infusions in patient 3.

times decreased towards the end of the period of chlormethiazole infusions as experience was gained in administration of the appropriate dose of chlormethiazole, and the doses administered were considerably lower than during the first few days.

Large changes in heart rate or blood pressure were not seen. The maximum decreases in systolic arterial pressure were 20–25% of those before the chlormethiazole infusion and probably not much greater than those which occur during natural sleep. Chlormethiazole may cause an increase in heart rate of about 20–40 beats/minute[1] but we did not observe this.

No problems in fluid or electrolyte balance were encountered that were not controlled easily by CVVHD.

Discussion

At the present time there is no ideal sedative for long term use in critically ill patients. Chlormethiazole provides good sedation with relative cardiovascular stability but one of its major drawbacks has been the large fluid load associated with its administration. CAVH, and more recently CVVHD and CAVHD, are used increasingly in critically ill patients with renal failure, and these techniques can be used to solve the fluid balance problems.

Relatively large doses of chlormethiazole were needed during the first 4–6 days and these were associated with a rapid recovery time. This represents cessation of clinical effect by redistribution. However, as the redistribution sites became saturated, the recovery times increased dramatically as termination of effect became dependent on metabolism. The dose must be reduced to match this change if acceptable recovery times are to be achieved.

Recovery time decreased later in the study period. This may represent the development of tolerance described with morphine,[11] fentanyl[12] and midazolam (unpublished observation). Alternatively clearance may have been reduced initially due to poor liver function, but may have increased subsequently as hepatic function improved; this phenomenon was observed during sedation with midazolam.[13]

Chlormethiazole infusions provided effective nocturnal sedation with cardiovascular stability over many nights in these three patients with renal failure. Once experience was gained with chlormethiazole, particularly in down-titration of the dose to avoid cumulation, it was found to be an effective and safe technique that provided smooth and consistent sedation in contrast to the previous difficulties experienced in providing adequate sedation with bolus doses of morphine and midazolam.

There is evidence from animal experiments that chlormethiazole might attenuate the cardiovascular and pulmonary instability associated with septic shock, which is a major cause of morbidity and mortality in the critically ill,[2] particularly in those with renal failure. This remains to be substantiated in man. Chlormethiazole may be used to provide both effective sedation and possibly prophylaxis against the adverse haemodynamic effects of sepsis. The relatively cheap price of chlormethiazole is a further advantage in the current climate of fiscal restraint.

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CASE REPORT

A plea for noradrenaline

M. E. STUART-TAYLOR AND M. M. CROSSE

Summary

Hypotension induced by nifedipine and chlorpromazine is discussed, together with the role of noradrenaline in the correction of this problem, which was resistant to other forms of therapy.

Key words

Complications; hypotension.
Calcium channel blockers; nifedipine.

Case history

A 52-year-old man was admitted for elective aortobifemoral bypass graft for occlusive vascular disease and was assessed 36 hours before surgery.

He had smoked 40 cigarettes per day for 40 years and had severe chronic obstructive airway disease, for which he received salbutamol and beclomethasone by inhaler. He was also prescribed bendrofluazide for hypertension. Psychiatrists were treating successfully with chlorpromazine a mixed psychiatric problem of schizophrenia and depression. He also took cimetidine and naftidrofuryl oxalate (praxiline). He had shortness of breath on exertion, with exercise tolerance limited to 0.25 mile on the flat, due to intermittent calf claudication. The ECG was normal.

The patient was found to have a blood pressure of 185/125 mmHg at the pre-operative assessment, but stated that he always became very hypertensive when admitted to hospital. However, the next day his blood pressure always returned to his usual level of 150/80. Examination of the records of his previous three admissions to hospital showed this statement to be correct. Instructions were therefore given for 4-hourly blood pressure measurements, no change in treatment, and, provided his blood pressure decreased to its usual level, surgery.

He was premedicated with temazepam 20 mg and metoclopramide 10 mg orally on the morning of surgery in addition to his normal medication. On arrival in the anaesthetic room his blood pressure was 150/95 mmHg. Peripheral venous, central venous and arterial lines were inserted under local anaesthetic. ECG, central venous (CVP) and arterial pressures were on continuous display and the patient was anaesthetised with midazolam 3 mg, fentanyl 250 μg, and vecuronium 8 mg. The blood pressure decreased over several minutes to 110/75 mmHg. Tracheal intubation resulted in an increase in blood pressure to 160/95 mmHg. Anaesthesia was maintained with 67% nitrous oxide in oxygen and end-tidal CO_2 was kept within normal limits. Volatile anaesthetic agents were avoided in view of the expected α-adrenergic block by chlorpromazine. The patient's blood pressure decreased progressively to 85/55 mmHg over the next 10 to 15 minutes. There was no response to intravenous infusion or to skin incision and, as the patient's right heart filling pressure was adequate, a dopamine infusion was commenced via the central venous line to reverse the hypotension. The initial dose was rapidly increased to 25 μg/kg/minute with no effect on the blood pressure. Incremental doses of 1:10 000 adrenaline were given over the next 10 minutes to a total of 1 mg. There was no change in arterial blood pressure which remained between 60/40 and 80/50 and there was a mild increase in pulse rate to 85 beats/minute.

A rapid assessment of possible causes of hypotension was made; this included air embolism, myocardial infarction, tension pneumothorax, cardiac tamponade from insertion of the CVP line, incorrect medication administered, poor calibration of pressure monitors and anaphylaxis. The first six possible causes were considered unlikely. However, the patient did appear flushed and vasodilated with bounding pulse; and in view of a recently administered antibiotic, an allergic reaction was considered possible. Chlorpheniramine 10 mg was given intravenously. The surgeon cross-clamped the aorta at this stage which also had no effect on blood pressure. A review of the patient's

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treatment card then revealed that nifedipine was commenced on the day after the pre-operative assessment.

Twenty millilitres of 10% calcium gluconate were given intravenously, followed by 15 mg methoxamine, both without effect. Finally a noradrenaline infusion was commenced at 0.03 μg/kg/minute and increased to a maximum of 0.19 μg/kg/minute which resulted in an increase in blood pressure to 110 mmHg systolic. Surgery progressed throughout this period. The noradrenaline infusion was gradually reduced over several hours after operation and the systolic pressure remained between 120 and 140 mmHg. Recovery thereafter was unremarkable.

Discussion

Calcium antagonists such as nifedipine are frequently employed to control hypertension, particularly pre-operatively, because of their relatively stable effect on the cardiovascular system. These drugs act by selective inhibition of transmembrane influx of calcium. The predominant effect of nifedipine is on the excitation/contraction coupling process in vascular smooth muscle which produces arteriolar vasodilation. Interactions of this class of drug with general anaesthetics are well documented in the case of volatile agents.[1] This case report illustrates a more unusual interaction, namely that between nifedipine and chlorpromazine. Phenothiazines have a dual mode of action on nervous tissue. Firstly, they block α-adrenergic neurones and secondly they inhibit the re-uptake of noradrenaline and 5-hydroxytryptamine in the central nervous system. We presume that these actions, combined with the calcium-blocking effect of nifedipine, produced marked vasodilatation, resistant to most of the vasoconstricting or inotropic agents used with this patient. It is reported in the literature that the antihypertensive effect of nifedipine is much more pronounced when it is taken together with cimetidine, as occurred with this patient.[2] Despite the ability of high-dose dopamine, adrenaline and methoxamine to stimulate α-receptors, their vasoconstricting effects were minimal, presumably due to the profound vasoplegia that resulted from α-blockade of chlorpromazine plus the direct vasodilating action of nifedipine. Calcium when administered on its own, produces vasoconstriction and an increase in systemic vascular resistance. However, in the presence of calcium antagonists calcium reverses myocardial depression but not vasodilatation. This helps to explain why the blood pressure did not respond to calcium given to this patient. Noradrenaline was the only effective vasoconstricting agent because of its highly selective stimulation of α-receptors.

This case demonstrates three facts. First, if one is faced with an unexpected change in the condition of a patient a methodical assessment of possible problems can highlight the cause. Second, one must be aware of the vasodilating properties of calcium antagonists, which are increasingly prescribed for hypertension and angina, particularly in combination with other vasoactive drugs. Noradrenaline has an important role to play in situations of undesirable hypotension and should not be confined to the realms of resuscitation or cardiac surgery. Finally before induction of anaesthesia the anaesthetist should check routinely the patient's treatment card, since medication may have been altered after the pre-operative assessment.

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Anaesthesia, 1989, Volume 44, pages 918–919

CASE REPORT

Unilateral subarachnoid anaesthesia

P. J. ARMSTRONG

Summary

The development of a unilateral block after subarachnoid anaesthesia is described. Other reported cases are discussed and the anatomy of subarachnoid space summarised.

Key words

Anaesthetic techniques, regional; spinal.
Complications; unilateral block.

Failure of subarachnoid blockade is a well recognised technical complication usually due to deposition of the local anaesthetic outside the subarachnoid space. A case that involves unilateral block after subarachnoid anaesthesia for Caesarean operation is reported.

Case history

A 26-year-old healthy primiparous woman (weight 75 kg, height 155 cm) presented for induction of labour at 42 weeks. Previous medical history was unremarkable and physical examination was normal. She had no back symptoms. She requested an epidural for pain relief after artificial rupture of the membranes. A 16-gauge end-hole catheter was inserted into the $L_{3/4}$ interspace using loss of resistance to air, and after a test dose of 4 ml 2% lignocaine plain, 8 ml 0.25% bupivacaine was given. A block of T_{10}–S_3 on the right side occurred with only a poor diffuse block on the left side of L_2–S_1. The mother was satisfied with this block and a continuous infusion of 20 ml 0.1% bupivacaine/hour set up. Over the next 8 hours she required two further top-up doses of 8 ml 0.5% bupivacaine. The block remained mainly unilateral with only poor pain relief on the left side.

She failed to progress and required a Caesarean section to deliver the baby; she wished to remain awake and the epidural block was insufficient for surgery, so subarachnoid anaesthesia was used. She was positioned on her right side and the epidural catheter removed. The subarachnoid space was easily located in the $L_{2/3}$ interspace with a 25-gauge spinal needle, and cerebrospinal fluid (CSF) flowed freely from the hub. Three millilitres of 0.5% heavy bupivacaine was slowly injected with the needle bevel horizontal. Repeated aspiration produced CSF during and at the end of injection. She was immediately turned onto her back.

She noticed a considerable increase in her motor and sensory block on the right side within one minute. She was unable to move her right leg after 3 minutes and sensation (pinprick and temperature) was blocked to T_3. No block was evident on the left side to either motor or sensation; there was a sharp loss of anaesthesia across the midline. Her arterial blood pressure remained unchanged. Twenty minutes after injection, her right side was totally blocked up to T_2 whilst there was no evidence of any block on her left side. It was decided to deliver the baby under general anaesthesia. Fifteen minutes after an uneventful operation she complained of soreness on her left side only and sensation testing showed her to be blocked to T_5 on the right with no block on the left. Anaesthesia on her right side gradually wore off over the next 2 hours. At no time did she notice any block on her left side. There were no postoperative sequelae.

Discussion

Unilateral block after epidural anaesthesia is well recognised with an incidence in obstetrical use of 1.5%[1] to 21%.[2] However, unilateral subarachnoid blockade is much rarer. Bozeman and Chandra[3] reported a similar event in a patient who had Caesarean section after a unilateral epidural. However, after subarachnoid injection with 10 mg amethocaine, the block only extended from T_6 to T_4 unilaterally. Injection of the amethocaine into the subarachnoid space would be expected to give a higher block and it is likely that the amethocaine was injected outside this space.[4]

Jenkins[5] reported a unilateral block in a 37-year-old

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male who had a vasovasotomy. Lignocaine 100 mg in 2 ml 7.5% dextrose with adrenaline was given in the $L_{3/4}$ interspace and the patient turned supine. A unilateral block developed within 5 minutes on the dependent side. Five minutes later a second subarachnoid injection in the same space with the unblocked side dependent was performed with a further 50 mg lignocaine; the needle was directed towards the unblocked side of the dura. He was kept in this position for 5 minutes when signs of a successful block occurred. However, it is possible that the lignocaine had insufficient time to diffuse across the subarachnoid space before the second injection was given.

Anatomically, many attachments of the arachnoid mater to the pia mater[6] exist that may interfere with diffusion of the local anaesthetic in the subarachnoid space. A distinct longitudinal midline dorsal septum, the septum posticum[7] crosses the arachnoid space from the dorsal midline of the arachnoid to the middorsal vein on the pia. It usually runs from the midcervical to the midlumbar regions and is fenestrated especially at its extremes. Strands also extend dorsally to the conus medullaris and even as far as the filum terminale. Lateral to this, on each side, exist the dorsolateral septa which extend from the dorsolateral root entry area on the pia to the arachnoid lateral to the roots. These septa are more irregular and fenestrated than the septum posticum. Laterally, the pia condenses to form the dentate ligaments that attach to the arachnoid between the root exits. Typically they stretch down to L_1.

The possibility exists that in some patients these septa may be complete and so cause divisions in the arachnoid space leading to incomplete or slow blockade. If the septum posticum was complete or if the dorsolateral septa surrounded the posterior roots then either unilateral or slow onset of anaesthesia could occur.

In the above case, after the subarachnoid injection of bupivacaine, there was a definite occurrence of a new block that remained unilateral for both sensation and motor blockade for the total duration of the block. Unfortunately, the patient refused follow up investigations to confirm the cause though it does appear likely that this was unilateral blockade after a subarachnoid anaesthetic.

Acknowledgment

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CASE REPORT

Anaesthesia in an infant with Joubert's syndrome

N. C. MATTHEWS

Summary

The presentation and management of a child with Joubert's syndrome is outlined with specific reference to the problems of opioid sensitivity and abnormal control of respiration.

Key words

Complications; Joubert's syndrome.

Joubert's syndrome is a very rare condition with few cases reported. It presents in the neonatal period with panting tachypnoea; followed by apnoeic attacks, jerky eye movements, rigidity and, in early infancy by ataxia and global developmental delay. This syndrome is caused by agenesis of the cerebellar vermis with cystic malformation of the brainstem. It is associated with Leber's Amaurosis (congenital retinal blindness) sufficiently frequently to suspect that the same autosomal recessive gene is responsible. Thus electroretinography is indicated in neonates with respiratory irregularities, particularly panting. The prognosis of Joubert's syndrome is poor with no survivors beyond the age of 4 years.[1,2]

Case history

S.D. was born at 38 weeks' gestation as an apparently normal vaginal delivery (birth weight 2.84 kg) and discharged home. He presented to his general practitioner at 3 weeks with vomiting after feeds. The general practitioner diagnosed a right inguinal hernia which was reducible.

He attended the casualty department at Birmingham Children's Hospital where he weighed 2.9 kg; the hernia was confirmed, and elective hernia repair was planned. An entry in the notes also described the child as abnormally jittery.

He came to hospital again 3 days later with an irreducible right inguinal hernia. Gallows traction was organised and papaveretum 0.5 mg given intramuscularly and again 2 hours later for hyperventilation, which was presumed to be due to pain. The hernia subsequently reduced spontaneously. He became more rigid with an increasing incidence of apnoeic periods which responded initially to stimulation. His respiratory rate decreased progressively to 15 with one episode of severe apnoea of at least 2 minutes duration, associated with cyanosis 6.5 hours after opioid administration. This responded immediately to naloxone 0.2 mg intramuscularly. Severe bradypnoea recurred and responded to naloxone 0.2 mg. He remained drowsy for a further 48 hours and was again noted to be jittery; he did not follow visual stimuli and exhibited irregular apnoea. The same day he was seen by a consultant paediatrician who suspected Joubert's syndrome. Subsequent CT scan revealed cerebellar hypoplasia and a cystic brainstem lesion. An electroretinogram, performed after an oculogyric crisis, showed a greatly reduced amplitude suggestive of retinopathy. The diagnosis of Joubert's syndrome was confirmed and he was discharged home with an apnoea alarm.

He returned one month later for repair of the right inguinal hernia. His mother recalled several apnoeic spells and many rigid episodes in the intervening period; he neither smiled nor followed visual stimuli. Admission weight was 5.1 kg. No premedication was given. Anaesthesia was induced with thiopentone 5 mg/kg followed by atracurium 0.5 mg/kg and a 3.5-mm Portex tube was inserted. Maintenance consisted of controlled ventilation of the lungs with 67% nitrous oxide in oxygen and 0.5–1.0% inspired isoflurane. An ilio-inguinal/ilio-hypogastric nerve block was done before surgery using 0.5 ml 0.5% plain bupivacaine. The procedure lasted for 35 minutes, and adequate spontaneous ventilation and movement resumed without the use of an anticholinesterase; the child was transferred to the Intensive Care Unit for observation and monitoring.

The patient was placed into a head box with 30%

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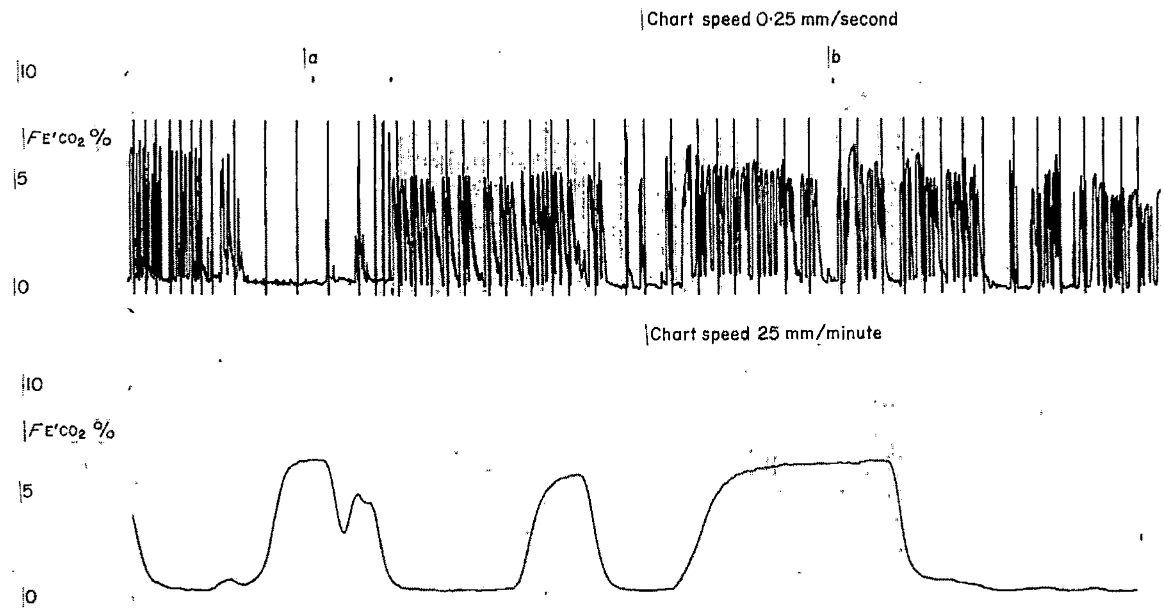

Fig. 1. Upper trace—end-tidal CO_2 (with calibration pulses of 8%) showing irregular apnoeas. Lower trace—demonstrating periods of sustained apnoea.

oxygen. End-tidal carbon dioxide was measured using a nasal catheter and the results recorded. The upper of the two charts show many apnoeic episodes; points a and b represent apnoeic periods of 120 and 10 seconds respectively. The highly irregular ventilation is demonstrated on the lower chart (see Fig. 1). These abnormalities persisted for some hours, but gradually lessened. The patient was transferred to the ward later the same day and discharged home.

Discussion

Infants with Joubert's syndrome may present a problem to the anaesthetist, because of medullary abnormalities with consequent abnormal control of respiration. They are unusually sensitive to opioids, which should be avoided where possible. Intermittent positive pressure ventilation is preferable to spontaneous ventilation, because of the apnoeic episodes and sensitivity to central nervous system depressants. Respiratory monitoring in a high-dependency environment is advisable after anaesthesia.

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Anaesthesia, 1989, Volume 44, pages 922–924

APPARATUS

A disposable device for patient-controlled analgesia with fentanyl

D. J. ROWBOTHAM, R. WYLD AND W. S. NIMMO

Summary

A disposable nonelectronic patient-controlled analgesia device was used to deliver fentanyl after upper abdominal surgery. Pain relief was satisfactory and plasma fentanyl concentrations were similar to those obtained by other workers who used electronic patient-controlled analgesia devices.

Key words

Pain; postoperative.
Equipment.

Patient-controlled analgesia (PCA) is used frequently for the relief of pain after surgery.[1] Several electronic infusion devices are now available but these machines are expensive and considerable capital investment is required if they are to be made available to all patients after surgery.

Travenol laboratories have developed a lightweight, nonelectronic disposable PCA system which is convenient for the patient and simple to operate.[2] We have assessed this device and measured plasma fentanyl concentrations after upper abdominal surgery. Results were compared with those of Gourlay and others[3] who measured plasma fentanyl concentrations during fentanyl delivery by an electronic PCA device (On-Demand Analgesia Computer, Janssen).

Methods

Eleven patients who had upper abdominal surgery were studied. Local ethics committee approval and informed written patient consent were obtained.

The anaesthetic regimen was standardised. Anaesthesia was induced with thiopentone 5 mg/kg after premedication with papaveretum 10–15 mg and prochlorperazine 12.5 mg. Fentanyl 200 μg and alcuronium 15–20 mg were given at induction and suxamethonium was used only if indicated clinically. Anaesthesia was maintained with enflurane 0.5–2% and nitrous oxide in 33% oxygen. Patients had artificial ventilation of the lungs to normocapnia. Supplements of alcuronium were given as required and neuromuscular blockade was reversed with neostigmine 2.5 mg, given with atropine 1.2 mg at the end of surgery.

Postoperative analgesia

Pain relief after surgery was delivered by the disposable Travenol PCA system. This consists of two components; a balloon reservoir constant-infusion device[4] which is connected by microbore tubing to a patient-control module worn around the wrist (Fig. 1). This was attached to the intravenous infusion line via a Cardiff nonreturn valve.[5]

The patient-control module contains a small bladder with a capacity of 0.5 ml (Fig. 2). The bladder fills in 6 minutes because the infusor used in this study delivers solution at 5 ml/hour. The patient presses a button on the face of the PCA device when analgesia is required. The button is recessed into the surround of the device to reduce the chance of inadvertent activation. The button, when pressed, releases a clamp and the contents of the bladder are delivered to the patient. There is no provision for the use of a constant-background infusion with this system.

The infusor was filled with fentanyl 2 mg made up to 50 ml with dextrose 5% to give a solution of fentanyl 40 μg/ml. This provided a bolus dose of 20 μg, lockout time of 6 minutes and maximum dose of 200 μg/hour. An adhesive label with a scale marked in centimetres was attached to the infusor in order to record hourly the movement of the

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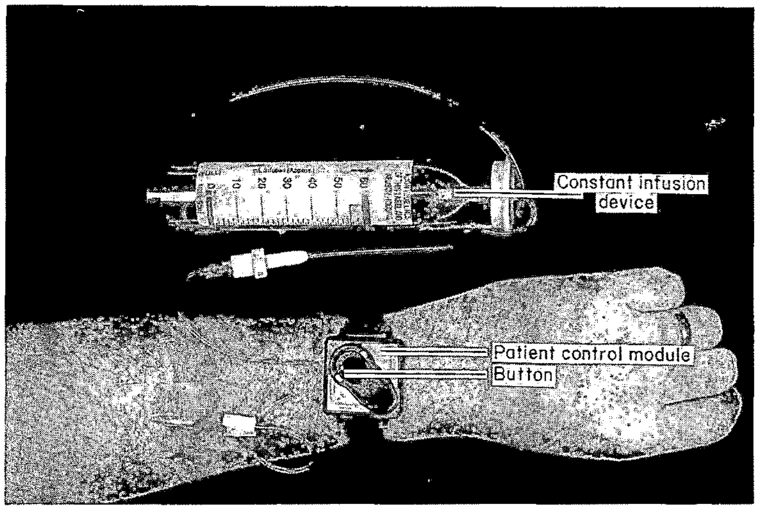

Fig. 1. Travenol constant infusion reservoir device connected via Luer lock to the patient-control module worn around the wrist.

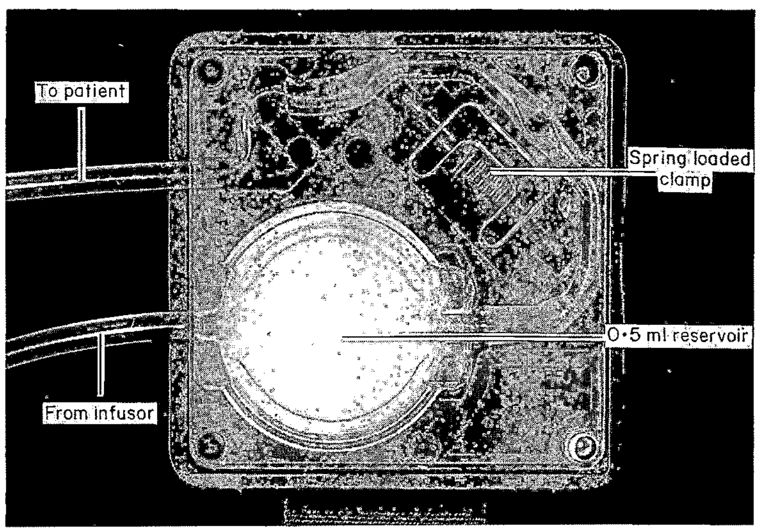

Fig. 2. Underside of the patient control module. The constant infusion device delivers solution into the 0.5 ml reservoir. The spring-loaded clamp is released when the button is pressed and the reservoir empties rapidly into the intravenous cannula.

infusor bladder. The study was stopped when the infusor became empty and intramuscular papaveretum was prescribed for pain relief thereafter.

Venous blood was taken at 12, 24, 36 and 48 hours after induction of anaesthesia. Plasma was separated and stored at −20°C until plasma fentanyl concentrations were measured by radioimmunoassay.[6] Pain was assessed by a 0–10 linear analogue scale at these times. The study was terminated if the infusor was empty at the time of assessment. Respiratory rate was measured hourly and patients were to be withdrawn from the study if respiratory rate decreased below 8/minute.

Results

Eleven patients (five female) were studied. Mean age was 56 years (SD 17) and mean weight 64.7 kg (SD 14.0). Upper abdominal surgery was performed on each patient (four

Table 1. Mean (SD) and ranges of plasma fentanyl concentrations at 12, 24, 36 and 48 hours (ng/ml).

	12 hours ($n = 11$)	24 hours ($n = 11$)	36 hours ($n = 9$)	48 hours ($n = 5$)
Plasma fentanyl (ng/ml)	1.4 (0.7)	1.4 (0.6)	1.2 (0.3)	0.5 (0.2)
Range	0.2–2.2	0.7–2.7	0.8–1.6	0.2–0.8

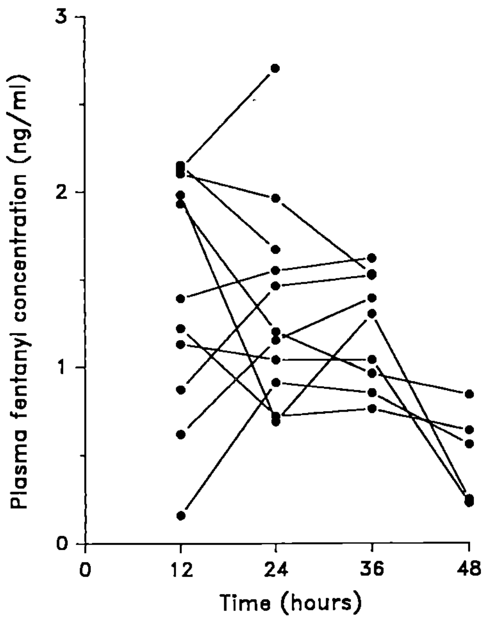

Fig. 3. Plasma fentanyl concentrations of each patient during fentanyl delivery by disposable PCA system.

Table 2. Median (ranges) pain scores at 12, 24, 36 and 48 hours. 0 = no pain, 10 = worst possible pain.

12 hours ($n = 11$)	24 hours ($n = 11$)	36 hours ($n = 9$)	48 hours ($n = 5$)
4 (2–5)	3 (0–5)	3 (0–4)	2 (0–3)

cholecystectomy, three gastrectomy, two antireflux procedure, one selective vagotomy, one oesophagomyotomy). No patient was withdrawn from the study.

Mean (SD) plasma fentanyl concentrations with ranges at 12, 24, 36 and 48 hours are shown in Table 1, and plasma fentanyl concentrations in each patient in Figure 3. The infusor had emptied in two patients (18%) at 36 hours, and at 48 hours in six patients (55%).

Median pain scores with ranges are shown in Table 2. No patient had a respiratory rate of less than 8/minute during the study, or required supplementary analgesia.

Discussion

Pain scores in our patients after upper abdominal surgery were low and no patient required analgesia supplementary to the disposable PCA device, which allowed a maximum hourly dose of 200 μg fentanyl.

Plasma fentanyl concentrations were similar to those found by Gourlay and others[3] using the On-demand Analgesia Computer (ODAC). These workers described minimum effective concentration (MEC) of fentanyl for pain-relief after surgery, a concept that implies constant analgesia when plasma opioid concentration is greater than MEC.[3,7] The mean MEC was 0.6 ng/ml (SD 0.25, range 0.2–1.2). We did not measure MEC, but plasma fentanyl concentrations in our study were greater than these values at all times. The maximum plasma fentanyl concentration in our study was 2.7 ng/ml. The patient's respiratory rate was 16/minute at this time.

One disadvantage of this system is that it is not possible to record accurately the number of boluses or the total dose administered to the patient. An approximate measurement can be made from the markings on the side of the infusor. Adhesive labels are available to facilitate this (Fig. 1). Such measurements, in our study, were made with the hourly respiratory observations.

In conclusion, after upper abdominal surgery, the Travenol disposable PCA device resulted in plasma fentanyl concentrations similar to those produced by an electronic PCA device. This device may provide PCA for many patients without the need for the investment in costly electronic pumps.

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Forum

Dose-response studies of atracurium, vecuronium and pancuronium in the elderly

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Summary

Dose-response curves were constructed for atracurium, vecuronium and pancuronium in elderly subjects in order to assess potency of these relaxants. The results were compared to data previously obtained for adult subjects using the same method. A single-dose method of potency determination was used in both studies. The results indicate no significant difference in the potency of these relaxants between elderly and adult subjects; the ED_{95}s were 249 and 226 µg/kg for atracurium, 43.1 and 39.6 µg/kg for vecuronium and 65.9 and 60 µg/kg for pancuronium respectively in the elderly and the adults.

Key words

Neuromuscular relaxants; atracurium, vecuronium, pancuronium.
Anaesthesia; geriatric.

Several workers have shown that the duration of action of pancuronium, even when used in single doses, is prolonged in the elderly.[1-3] The durations of action of single doses of vecuronium and atracurium, on the other hand, have not been reported to be prolonged in such patients.[4] It is not clear whether this is due to different sensitivities to these relaxants in the elderly. There is, moreover, no information available on the potency of atracurium in the elderly. The present study was designed to assess the potency of the three relaxants in the elderly and to compare the results with those obtained previously in healthy adult subjects.[5-7]

Methods

Ninety-one elderly patients of ASA grades 1 or 2 over the age of 65 years were admitted to the study with their informed consent and Ethics Committee approval.

Anaesthesia was induced, after premedication with oral diazepam, with thiopentone 3–5 mg/kg and fentanyl 50–100 µg/kg and maintained with 67% nitrous oxide in oxygen, and further increments of fentanyl 25–50 µg/kg as required. Ventilation was adjusted to maintain an end-tidal carbon dioxide concentration of 4.5–5.0%. The ulnar nerve was stimulated percutaneously at the wrist with supramaximal square wave stimuli of 0.2 msec duration at 0.1 Hz, and the resultant force of *adductor pollicis* contraction measured and recorded using a force displacement transducer and a neuromuscular function analyser (Myograph 2000, Biometer). Stabilisation of the control twitch height was followed by random allocation to receive a single bolus dose of atracurium 100, 150, 200 or 250 µg/kg, vecuronium 20, 30, 40 or 50 µg/kg or pancuronium 20, 30, 40, 50 or 60 µg/kg. The maximum twitch height depression and the time taken to attain it was recorded in each case.

The data were subjected to arc-sine transformation as described by Armitage[8] for responses that involve the extreme end-points (0 and 100%) on the dose-response curves. Dose-response curves were constructed after regression analysis, and the respective ED_{50} and ED_{95} (doses required for 50 and 95% depression of the twitch height respectively) derived. The slopes of the dose-response curves and the ED_{50} and ED_{95} were compared using *t*-tests with results obtained previously in adult subjects using the same method.[5-7]

Results

There were seven patients in each dose group, and each group was comparable with respect to age and weight (Table 1). The mean age of the groups from the previous studies in adult patients was also comparable, but, as expected, significantly lower in comparison to the respective elderly groups.

The dose-response curves obtained for the three relaxants in the elderly are shown in Figure 1 along with those obtained previously in adult subjects. The dose-response curves did not differ in their slopes between the elderly and adult patients for each relaxant. The calculated ED_{50} and ED_{95} are given in Table 2. The ED_{95}s for atracurium, vecuronium and pancuronium in the elderly patients were 249, 43.1 and 65.9 µg/kg respectively. These were not significantly greater than 226, 39.6 and 60 µg/kg in the adult subjects. There was no difference in the ED_{50} values.

The maximum block produced by each increment of the muscle relaxants and the time taken to attain it showed no significant difference between the elderly and adults.

Discussion

The present study demonstrated no significant difference in the potencies of atracurium, vecuronium or pancuronium between adults and the elderly. Comparison was made with

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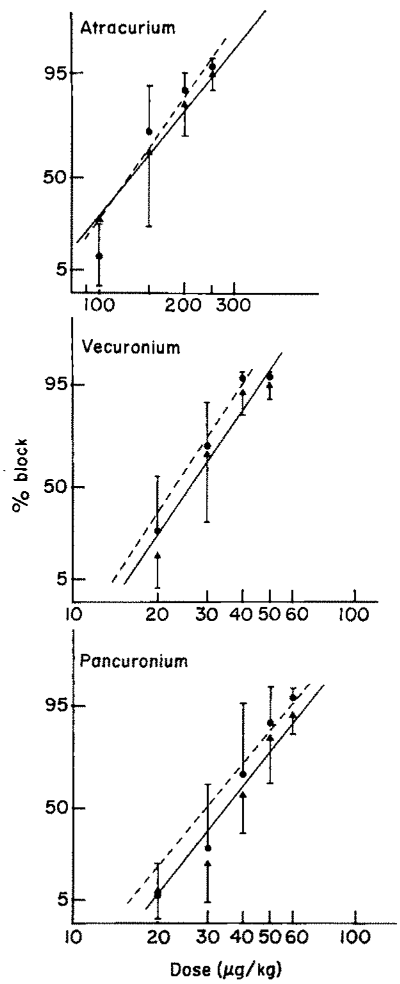

Fig. 1. Dose-response curves for atracurium, vecuronium and pancuronium in the elderly ▲——▲ and adult patients ●---●. Doses on the x axis are on the log scale and responses that follow arc-sine transformation are on the y axis. Data for adult patients from previously published work (references 5–7).

control data previously obtained, although these data were objective and were obtained using the same methods. There was, in addition, no significant difference in the time taken to attain the maximum block with each dose. Our results confirm the previous report about the potency of vecuronium.[9] Duvaldestin *et al.*[2] reported similar ED_{50} values for pancuronium in the elderly and younger patients but they did not quote any ED_{95} values. Their study, however, showed the duration of action of pancuronium to be prolonged in the elderly. This was attributed to an increased elimination half-life and reduced clearance in the elderly, although a more recent study showed no significant differences in the kinetics of either pancuronium or vecuronium between elderly and adult subjects.[10]

Table 2. ED_{50} and ED_{95} (μg/kg) values for elderly and adult patients (95% confidence limits in parentheses). Data for adult patients from previous studies (references 5–7).

		Elderly	Adult
Atracurium	ED_{50}	137 (121–154)	126 (115–137)
	ED_{95}	249 (210–289)	226 (207–246)
Vecuronium	ED_{50}	25.7 (23.3–28.4)	23.1 (20.6–26.0)
	ED_{95}	43.1 (38.9–47.7)	39.6 (35.7–43.1)
Pancuronium	ED_{50}	36.1 (33.3–39.2)	30.0 (24–36)
	ED_{95}	65.9 (57.0–76.2)	60.0 (48–72)

It was reported that the requirements of vecuronium for steady state relaxation are lower in the elderly when the drug is used by continuous infusion.[11] This is different from the results obtained using only single doses,[4] which may be due to kinetic factors. The kinetics of vecuronium after administration by continuous infusion are not known. There is no information for atracurium either for potency determination or for kinetics in the elderly, although published reports do not suggest any significant prolongation of the clinical effect.[4,12]

The technique of potency determination was shown to be important, particularly with the introduction of atracurium and vecuronium. A single-dose method of potency determination yields more accurate results for these relaxants,[5,6,13] whereas no significant differences exist between single and cumulative dose techniques in pancuronium.[7,14]

The dose-response curves for the elderly were slightly to the right of the curves for the adult subjects in the present study, and suggest decreased potency of muscle relaxants in the elderly. Such a shift in the dose-response curves was also demonstrated by Choi *et al.*[3] for pancuronium. Metabolic and histochemical studies in man show an increase in slow fibre component of muscles with age and these may be more resistant to the effects of muscle relaxants.[15]

In conclusion, it appears that kinetic factors, rather than sensititivity to muscle relaxants are responsible for the prolonged duration of action of some muscle relaxants in the elderly.

Acknowledgments

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Table 1. Age and weight of patients. Adult group data are from previous studies (references 5–7).

	Age, years (SD)		Weight, kg (SD)	
	Elderly	Adult	Elderly	Adult
Atracurium	75.0 (6.5)	40.0 (18.2)	68.9 (12.2)	65.0 (13.0)
Vecuronium	74.3 (6.7)	38.0 (6.9)	62.2 (11.2)	67.0 (8.5)
Pancuronium	74.0 (7.4)	44.0 (15.0)	63.3 (10.7)	66.0 (11.4)

$p<0.05$ for age between elderly and the adults for each relaxant.

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Anaesthesia, 1989, Volume 44, pages 928–941

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An unusual emergence after total intravenous anaesthesia

We would like to report another case of postoperative hallucinations[1,2] after a total intravenous anaesthetic with propofol, fentanyl, and atracurium.

The patient was a 27-year-old, healthy, married woman who had a propofol anaesthetic for excision of a cold nodule in an otherwise normal euthyroid gland. Premedication consisted of midazolam 7.5 mg orally 90 minutes before operation. Induction was with fentanyl 0.2 mg and a propofol bolus of 1 mg/kg, followed by an infusion of propofol starting with 10 mg/kg/hour. Infusion rate was stepwise reduced to 6 mg/kg/hour within 20 minutes and fentanyl 0.1 mg boluses were administered according to

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clinical needs up to a total of 0.4 mg. Tracheal intubation and ventilation of the lungs with oxygen-enriched air were facilitated by intermittent injection of atracurium. The intra-operative course was uneventful. She seemed dysphoric and refused to communicate with the staff on regaining consciousness. She was noted to be unhappy, restless, and distressed in the recovery room. She asked to see a senior nurse officer in the evening, about 7 hours after the end of the anaesthetic. She revealed that she was sexually harassed in the recovery area by a man whom she could not identify because she was not able to open her eyes. She described the panic and sheer terror of being caressed for approximately 5 minutes while unable to shout or communicate her distress to the recovery staff.

The patient was entirely convinced that she was not hallucinating and her experience was absolutely real. Our recovery ward, with 10–12 holding bays, is permanently staffed by three qualified female recovery nurses, all of whom stated independently that for the time our patient was kept in recovery there was nobody in her vicinity except the female nurse who looked after her.

She considered the possibility of a drug-induced post-operative hallucination the next day, but remained unconvinced that it was only an imaginary experience.

Two points are worth mentioning apart from the sexual nightmare during recovery: the rather unusual emergence from a propofol anaesthetic with dysphoria and restlessness and prolonged impairment of normal mental performance; the inability to open the eyes, even in a situation of direst distress. We have observed similar problems with eye-opening in a group of ENT patients after the anaesthesia technique described above, although all of these patients were communicative and orientated in space and time.

Whether propofol was responsible for, or facilitated the occurrence of, such a devastating experience cannot be determined because fentanyl and midazolam might have contributed to this critical event. Clearly close post-operative supervision during emergence from total intravenous anaesthesia is mandatory. It may be helpful in light of the permanent threat of allegations of sexual impropriety,[3] to ensure that a female third party is always present when the above mentioned drugs might interfere with normal perception.

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Thoracic epidurals and general anaesthesia

Dr Bromage's criticism (*Anaesthesia* 1989; **44:** 445) of Gough *et al.'s* placement of thoracic epidural cannulae in anaesthetised patients, and their reply were very interesting. Dr Bromage was to me, as to many anaesthetists 15 years and more on each side of my age, a shining beacon to be followed through the epidural learning process. Whatever he wrote was accepted, like the tablets on the mount, as the wisdom from on high. For this reason, I feel constrained to write to you on this subject because, for once, I think that my god is, if not wrong, at least too severe.

A person of Philip Bromage's stature and experience must always weigh very carefully all that he says, lest someone feels that what he has written is a licence to malpractise. His caution and personal advice to myself have made me aware, as never before, of the dangers which lie in wait for any anaesthetist, but particularly, as he said, for those who practise local anaesthetic techniques, especially those close to the spinal cord.

That said, I do not feel that his criticism of thoracic epidural cannulation in sleeping patients was completely reasonable. My practice has included placement of at least 3–400 thoracic epidurals over the last 10 years without cord puncture. The nature of my technique, which is to use a lateral approach together with a negative pressure test in a patient who is hyperventilating should, I hope, diminish the chance of hitting the cord. But the angle of insertion is at least as important. The angulation of the spinous processes in the upper thoracic spine is such that one must approach at something like 25–30 degrees to the skin. One would have, at this angle, to advance the (blunt) Tuohy needle some 2–3 cm beyond the ligamentum flavum, and puncture the dura mater without realising it, before making contact with the cord itself. No anaesthetist should embark upon thoracic epidurals without considerable experience of the lumbar approach, and this experience should have weeded out those who are that clumsy.

'Blind invasion of the spinal canal even in the most skilled hands, can never be as safe asleep as awake', does not convince me either. My commonest complication of epidural anaesthesia is dural puncture. A proportion of these in awake patients was the result of the patient suddenly jumping as the needle touched a spot which had not been anaesthetised by the local anaesthetic infiltration. A lancinating injury of the cord could, indeed, result if this were to happen where the needle was close to the cord.

The condemnation by so eminent a person as Dr Bromage may imperil what I consider to be a humane and safe procedure. Put on the personal level, I should be happy to submit my anaesthetised self to a thoracic epidural at the hands of an experienced anaesthetist.

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An alternative to the oesophageal detector device

Unrecognised oesophageal intubation is still a major cause of patient injury and litigation against anaesthetists[1–3] so any steps which could reduce this hazard should be evaluated. Drs Williams and Nunn (*Anaesthesia* 1989; **44:** 412–4) describe an ingenious method which does not require any external energy and is inexpensive. However, the oesophageal detector device does not quantify anything and relies on the observations and experience of the user. Drs Williams and Nunn's assessor was an anaesthetist and it would be interesting to repeat the study using nonanaesthetic personnel. The reason for suggesting this approach is related to tracheal intubations performed outside the oper-

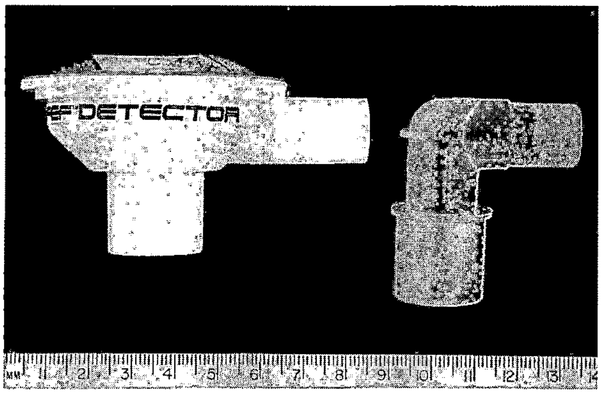

Fig. 1.

ating theatres, often in less than ideal circumstances, and by inexperienced operators. A method to measure carbon dioxide directly rather than by inference would be advantageous on these occasions.

A pH-sensitive chemical indicator which could detect CO_2[4] was developed in 1988. The indicator was enclosed in a disc housing and could be placed in the gas stream between a tracheal tube and anaesthetic breathing system. Recently, we described[5] the first commercial application of this technology, the FEF end-tidal CO_2 detector (Fig. 1) manufactured by Fenem Airway Management Systems, 84 Williams Street, New York 10038, USA.

Figure 1 shows the detector with a standard angle piece for comparison. The device is made of white plastic and contains the chemical indicator on a mesh in its upper part; the dead space is 38 ml and the inlet and outlet ports standard 15 mm. There is a round clear window on the upper part so that the colour of the mesh can be seen. The detectors are packed individually in gas tight foil bags and their shelf-life unopened is 15 months. The current unit cost in the USA is $15. When attached to a tracheal tube the initial colour of the indicator is purple. However, if gas that contains carbon dioxide passes through the device, either from spontaneous or artificial ventilation, the purple colour fades immediately to pale yellow. Fresh gas that contains no carbon dioxide will restore the original purple colour. The changes are sufficiently fast to allow a breath by breath response in adults. It is meant for immediate use to determine correct placement of a tracheal tube, but we have left the devices in the anaesthetic system and found that while they are not affected by nitrous oxide or volatile agents the response of the indicator fades gradually after a few hours. There appear to be no problems with regard to water vapour or changes in ventilatory pattern.

We suggest that when the challenge posed by Drs Williams and Nunn to carry out widespread evaluation of their device is taken up, similar consideration be given to the FEF detector.

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Comments on the oesophageal detector device

Drs Williams and Nunn's prospective trial on 100 patients of the oesophageal detector device (ODD), (*Anaesthesia* 1989; **44:** 412–4) was interesting. They used Ellick's bulb as the aspirating device instead of the original manual aspiration using a 60-ml syringe,[1] for the sake of simplicity, and to avoid confusion, I will refer to them as ODD Mark 2 and ODD Mark 1, respectively. There were no false positive results in the detection of placement of the 'tracheal' tube, but the authors' assessors had the benefit of using the ODD Mark 2 in both the oesophageal and tracheal placed tubes. This makes the decision, as to which tube was located in the trachea or oesophagus, much easier to determine. One does not have this luxury of choice between two tubes in the clinical situation.

Most anaesthetists after tracheal intubation connect the patient to a breathing system and ventilate the patient's lungs to check tube placement. This is almost a reflex action. Williams and Nunn found delayed refill of Ellick's bulb in 16 cases of oesophageal intubation. What proportion of the oesophageal intubated patients have delayed refill, or perhaps even instantaneous refill, if the tube in the oseophagus were first connected to a breathing system and given one or two tidal volume inflations? There is no mention in their study that the ODD Mark 2 must be used *before* gas is introduced down the tube.

The clinical situation was simulated in the original clinical trial of the ODD Mark 1 by the introduction of approximately 2 tidal volume compressions of the reservoir bag of the breathing system before the assessor, who had not witnessed the anaesthesia, used the ODD Mark 1 on the left-hand-side placed tube which could be in the trachea or the oesophagus. There were no false positive or negative results.

I have now personally used the ODD Mark 1 in over 2000 patients without any incidence of false positive results although there were only 27 cases of inadvertent oesophageal intubation. There were 25 cases of tracheal intubations (16 with the cuff inflated and, latterly, nine with the cuff deflated) where one could aspirate gas, typically between 20 and 40 ml, and then encounter some resistance followed in some instances by gas again. This 'resistance' is quite different from the marked resistance encountered during aspiration of a tube in the oesophagus. One has to accept that there will be a small percentage of cases where the bevel of the tube may be temporarily occluded by the tracheal mucosa, and this seems to occur more often in obese patients, when there may be some degree of distortion of the trachea; in endobronchial intubation, where there is distortion of the tube in the narrower bronchus, or when aspiration is done 'enthusiastically', this causes suction which encourages adherence of the bevel of the tube to the tracheal mucosa. One must also remember tube obstruction can be caused by kinking or presence of material in the tube. It is interesting, but not surprising to note that resistance during an aspiration test in a tube in the trachea has never occurred in tubes which have the

Murphy eye.

Drs O'Leary and colleagues[2] stated in their evaluation of a similar device to the ODD Mark 1 that it is essential the cuff of the tube be deflated during aspiration because of the theoretical risk of hypoxia and bronchial collapse. It is hard to imagine that 30–40 ml aspirated from a total resting adult lung volume of approximately 2500–4000 ml causes significant hypoxia and (or) bronchial collapse. It would seem more prudent in the patients with high risk of gastric aspiration to secure the airway by inflation of the cuff in addition to cricoid pressure until tube placement is confirmed.

The ODD Mark 1 has been used successfully in a recent preliminary trial in children using uncuffed tubes.[3] Further wider evaluation of its use in children would be useful.

The following are guidelines for the use of the ODD Mark 1 in adults. Check that the device is airtight, including the fitting to the tracheal tube. Apply constant slow aspiration to avoid the suction effect and prevent mucosal damage. The tracheal tube is correctly placed in the trachea if 30–40 ml gas is aspirated without resistance. If gas is initially aspirated and then resistance is encountered, confirmation of tracheal tube position, if needed, should proceed as follows. Retract the tracheal tube 0.5–1 cm and rotate it partially. This takes the tube out of the bronchial position if it is too long and also changes the orientation of the bevel. This manoeuvre can be done in one motion. Gas will be aspirated if the tube is in the trachea whilst it will make no difference if the tube is in the oesophagus. The ODD Mark 1 can be used before or after ventilation of the patient and in patients with high risk of gastric aspiration. It is recommended that the cuff of the tube be inflated before the test. Continuous cricoid pressure should be applied until tube placement is confirmed. Another different use of the ODD Mark 1 is to use it as a safe conduit for gastric contents. The ODD Mark 1, without its plunger, attached to the oesophageal tube can act as a safe conduit for gastric contents to a location away from the head, if the oesophagus is inadvertently or deliberately intubated. This will be particularly useful in patients with a full stomach who are possibly difficult to intubate and where repeated attempts at intubation may be needed.

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A reply

We are glad of the opportunity to respond to Dr Wee's very helpful comments on our modification of his oesophageal intubation detector device (*Anaesthesia* 1988; **44:** 412). There can be no doubt that making a direct comparison of one tube in the trachea with another in the oesophagus aids the recognition of oesophageal intubation. Nevertheless, in 98% of our patients the instantaneous refill of the Ellick's evacuator left us in no doubt at all that the tube was in the trachea. We have never seen instantaneous refill from the oesophagus. However, Dr Calder and his colleagues have reported a case of failed refill from a tracheal intubation (*Anaesthesia* 1989; **44:** 705).

Dr Wee also draws attention to the possibility of refill from an oesophageal intubation if an attempt were made to ventilate the lungs through a tube erroneously placed in the oesophagus. He rightly says that our test should be undertaken *before* any gas is introduced through the tube. This is, in fact, our normal practice and presents no problem since the test only takes 3 seconds, usually a negligible delay in the initiation of ventilation.

The essence of our method is its speed, simplicity, availability, negligible cost and high success rate. However, nothing in medicine is fool-proof. Any new device must be carefully monitored, as in the case of new drugs, but this is useless if the problems are not reported.

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Oesophageal detector device

Drs Williams and Nunn (*Anaesthesia* 1989; **44:** 412–4) are to be congratulated on their report of the use of an oesophageal detector device.

Any procedure that reduces the possibility of incorrect placement of the tracheal tube is a considerable contribution to the increase in patient safety.

However, I note that instantaneous refill of the rubber bulb attached to a tube placed in the oesophagus did not occur in a single instance.

It is a not uncommon observation, if the stomach were inflated inadvertently by the use of a mask prior to attempted intubation, that the reservoir bag will refill with an instantaneous 'rebound' normally associated with correct inflation of the lungs. I suspect that in these circumstances the oesophageal detector will not confirm that the tube is in the oesophagus.

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A reply

We thank Dr Mathias for his interest in our device and yourself for the opportunity to reply.

Sadly, our device does nothing to reduce the possibility of misplacing a tracheal tube; it merely increases the chances of detecting such an event.

All but two of our patients were intubated with the help of non-depolarising neuromuscular blocking agents and it is not unreasonable to assume these were all ventilated by mask to some extent before intubation. Despite this we did not observe a single instance of instantaneous refill from the oesophagus. Perhaps more widespread use will reveal whether Dr Mathias' fears are justified.

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Propofol in acute porphyria

The use of propofol in patients with acute porphyria remains controversial.[1–3] Hodkinson[2] stated that in most of the clinical reports, the total dose of propofol was less than 500 mg and the urinary porphyrins were not measured.

We recently published two cases of acute porphyria where propofol was used without adverse effects.[4,5] The first case concerned a 21-year-old woman with familial intermittent porphyria. She had surgery for a right ear cholesteatoma. The total dose of propofol administered was 740 mg for 110 minutes. The postoperative recovery was rapid and without complication. Screening for urinary porphyrins was carried out before and after the surgical procedure. No significant modification was noted in the porphobilinogen, uroporphyrin and coproporphyrin. The second case concerned a 52-year-old woman with acute intermittent porphyria. In this family, 27 people affected were detected, of whom four died from complications directly connected to the disease. The patient underwent a total hysterectomy for a fibroma of the uterus under general anaesthesia with propofol, fentanyl and vecuronium. A total dose of 800 mg propofol was given over 190 minutes. The postoperative period was uneventful. The urinary porphyrins were not measured due to absence in the change of coloration in the urine. These two cases confirm the view of McNeill *et al*[1] that propofol may be safe in acute porphyria.

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Unusual reaction to diclofenac

This is a report of an unusual reaction to diclofenac ('Voltarol', Ciba-Geigy) injection.

A previously healthy 41-year-old Caucasian female was admitted for bilateral first metatarsophalangeal osteotomy and fusion. She weighed 54 kg and had no known allergies; she was anaesthetised for hysterectomy 3 years before, with no untoward sequelae.

She was premedicated with lorazepam 2.5 mg and domperidone 10 mg orally. Anaesthesia was induced through a 20-gauge indwelling needle on the dorsum of her left hand with propofol, alfentanil, and droperidol, and continued with isoflurane in oxygen. An intramuscular injection of diclofenac (75 mg in 3 ml) was given into the outer aspect of the left thigh immediately after induction.

Bilateral leg tourniquets were applied below the injection site. The procedure, which lasted 30 minutes, was concluded uneventfully.

On the first day after operation she reported, at the site of the injection, a painless blister about 1 cm in diameter with a central flat area; this burst on the second day. On the third day the lesion consisted of a hard black scab approximately 1 cm in diameter surrounded by a small (5-mm wide) zone of induration. The lesion was treated expectantly and remained unchanged until her discharge 7 days later.

Four weeks later she reported that her general practitioner had advised her to remove the scab and that the lesion was 1 cm deep and that she had been advised to pack it. One week later the lesion was 1 cm in diameter and 1 cm deep. Cultures showed coagulase-positive staphylococcus sensitive to flucloxacillin. Treatment was started with this and she was referred to a plastic surgeon who excised the site with primary repair.

The histology report was on 'a piece of skin, ellipse 2.1 × 1.1 cm. There is a small central defect and a sinus leads from this into the subcutaneous fat, where it is surrounded by dull yellow tissue. The sinus is surrounded by granulation tissue and leads to an area of panniculitis and fat necrosis with numerous foamy phagocytes and occasional giant cells.'

Serratrice[1] evaluated 10 167 cases and reported six (0.05%) abscess formations and three (0.02%) necrosis as part of a larger group of 744 (7.3%) showing local intolerance, principally (5.6%) pain at the injection site. Baroni[2] found 27 patients out of 1873 (0.01%) who showed reaction at the injection site, but did not specify the nature of the reaction.

This side effect does not appear in the UK literature and the incident was reported to the Committee on Safety of Medicines.

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General anaesthesia and the presence of the spouse

The letter from Freeman *et al.* (*Anaesthesia* 1989; **44:** 618) prompts me to write in support of the junior anaesthetist concerned, and I believe he (she) should be commended for the correct decision. A similar situation arose for me some years ago: I performed a spinal anaesthetic with 0.5% plain bupivacaine with the husband present in theatre. This spread rapidly to give a T_5 level for analgesia to pinprick and the operation started. However, the patient was unable to tolerate any handling of the peritoneum and asked to go to sleep. The husband then wanted to remain and watch his baby being born. The obstetrician had no objection, but left the decision to me and I agreed.

The procedure was uneventful and after an explanation of events the delighted father left theatre with his new baby accompanied by a midwife.

Despite my policy of usually having the partner present during induction of spinal or epidural anaesthesia I found the induction of the general anaesthetic stressful.

We ought however to think rationally about this situation. There is great emphasis today on childbirth as a shared experience and I see no reason to deny a father the right to see his baby being born even if the mother does not want to be awake. The policy in some obstetric units is that fathers are admitted to theatre once the mother is prepared and draped and ready for surgery under regional block. There is absolutely no reason why this policy cannot be followed for Caesarean sections under general anaesthesia, with the father leaving theatre shortly after delivery, with his baby.

However, I believe this easy-to-institute policy is hypocritical. We expect our obstetricians to perform under the gaze of a partner's sometimes quite professional eye; nurse, doctor, vet, butcher to name but a few! What we expect of our colleagues we too should be prepared to undertake.

Provided an adequate explanation of the procedure is given (no bad thing in these litigious times) I do not forsee any problems over and above what may happen in my normal regional anaesthesia practice. It is reassuring to note we can expect the full support of the defence organisations if the partner does interfere and causes harm to anyone.

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I.F. RUSSELL

We read the letter from Dr Freeman and colleagues (*Anaesthesia* 1989; **44:** 618) with a sense of *déjà vu.* Our patient also required an emergency Caesarean section because of fetal distress. Delivery of the baby was interrupted by the crash of theatre doors as the patient's husband forced his way in. His manner was both threatening and abusive and he refused all requests to leave. We attempted to enlist the presence of a security guard but without success. The staff completed the operation without mishap in an atmosphere of increasing menace and threat of assault. Nevertheless, such extreme distraction at a critical time clearly constituted an immense safety hazard to the patient and her baby.

It is fashionable to consider limitation of access by spouses under any circumstances to be a denial of freedom. We must refute this view and uphold the principle that a husband may only enter theatre at the discretion of the staff. Otherwise, we must meekly accept an increased risk to our patient.

When a passenger forcibly enters the flight-deck of an aircraft, he breaks the law. Likewise, a publican may legally refuse entry to any customer whom he believes is liable to cause trouble (no matter if his wife is sitting in the saloon bar!). We consider that operating theatre staff deserve at least equal consideration.

Departments may decide, as we have done, to formalise their view in an information document handed to all obstetric patients. A stated policy may reduce the number of such incidents, but there will remain a hard core of people who will flout the rules. Staff can only defuse the tension in this event, if there is ready access to a credible, round-the-clock, hospital security presence.

Will our masters wait for the inevitable catastrophe to occur before deciding to act?

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R. GADELRAB
J.G. ALLEN

Analgesia for manipulation under anaesthesia after total knee replacement

A few patients require manipulation under anaesthesia (MUA) after total knee replacement (TKR) operations to achieve the goal of 90° active flexion before discharge. The use of MUA seems to depend mainly on the preference of the individual surgeons, from its occasional to routine use in all patients.[1] The anaesthetic technique involves no more than a hypnotic dose of a suitable induction agent with or without a short-acting muscle relaxant and oxygenation.

What is remarkable, however, is the excruciating pain experienced by some patients during recovery from anaesthesia. It is not unusual, despite narcotic premedication, to employ up to 40 mg papaveretum intravenously for analgesia. When this had failed to provide relief, in one patient, a femoral nerve block was performed with 20 ml plain bupivacaine 0.5%, in a desperate attempt to control the pain. We were as astonished as the patient by the dramatic relief provided by this block.

Subsequently, two patients presented with bilateral TKRs for manipulation. A femoral nerve block (FNB) was performed on one side to assess its effect with the patients. Both patients recovered in agony, the pain was emphasised by the effective analgesia on the blocked side. A femoral nerve block was quickly performed on the painful leg: there was dramatic and complete relief in both patients.

The vigour with which manipulation is performed determines the intensity of pain as well as the extent to which adhesions are broken down. But it is the stretching of the quadriceps femoris, often fixed in painful spasm before operation, that is responsible for the excruciating pain after manipulation. Relaxation of this muscle after an FNB is the most likely explanation for the effective analgesia in these patients since a FNB alone would be insufficient to block nociception from the operated knee. A 3-in-1 block[2] is an unlikely explanation, because 10 ml bupivacaine was just as effective as 20 ml, but the analgesia was short lived.

We now routinely perform FNBs, and would recommend this for analgesia in all patients for TKR, or manipulation following TKR. It is neither likely to be effective, given the extent and painful nature of the manipulation, nor justified as a sole anaesthetic. Should a general anaesthetic be contraindicated, a subarachnoid or an epidural block is a suitable alternative.

We have since used this technique in 15 patients with excellent results. All patients achieved the required mobility at discharge without further complication or manipulation. Remarkably, once analgesia was effective in the immediate postoperative period, only one patient requested additional analgesia in the ward.

A further advantage of the FNB is that it allows the immediate and pain-free use in recovery, of the continuous passive motion (CPM) device for its many beneficial effects.[1,3]

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References

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Bed rest after lumbar puncture is obsolete

Prophylactic bed rest after lumbar puncture (LP) has been common practice for many decades. Cook, Davies and Beavis in their study (*Anaesthesia* 1989; **44:** 389–91) show that staying in bed does not prevent postlumbar puncture headache (PLPH) after spinal anaesthesia. Therefore they conclude that after spinal anaesthesia patients should be mobilised as soon as possible and I completely agree with their conclusions. They claim, however, to be the first to demonstrate this in a prospective trial, but that is not the case.

Carbaat and van Crevel demonstrated that bed rest only postponed PLPH after diagnostic LP, but that it did not lessen its incidence.[1] Studies since of different patient populations all support their findings. There were three studies investigating PLPH after LP for spinal anaesthesia.[2-4] One study investigated PLPH after LP for diagnostic purposes[5] and three studies investigated PLPH after LP for lumbar myelography or run-up cervical myelography.[6-8] Preventive bed rest was successful in none of these studies.

Therefore early mobilisation is recommended after LP and there is no longer any justification for the prescription of bed rest after LP performed for any of the indications named above.

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A reply

Thank you for the opportunity to reply. We agree that the literature overwhelmingly supports the view that bed rest does not prevent postspinal headache. However, we disagree when he says that we claimed to be the first to demonstrate this in a prospective trial. What our article said was that, when the trial was begun; 'no well-controlled prospective trial has been performed in surgical patients to validate these results' (that is, the results of Carbaat and Van Crevel).

It is true that since then a well-controlled study on *surgical* patients has been published on this subject by Andersen *et al.* The stimulus to perform our study was our desire to confirm in *surgical* patients what had been reported in the nonsurgical population.

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M.J. DAVIES
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Auriculo-emetic reflex?

The article (*Anaesthesia* 1989; **44:** 110–7) suggests that there is a central oculo-emetic reflex pathway which possibly accounts for the high incidence of vomiting after squint correction. This, the authors explain, may be due to maintained stress on the musculature of the squint-corrected eye, which stimulates the emetic centre and promotes vomiting.

It is not in my experience only the ocular musculature which may cause an emetic reflex, bat-ear correction may also produce a very high incidence of vomiting in children. The mechanism is not clear but if the nerve supply to the ear is blocked before correction, the incidence of vomiting is considerably decreased.

In a pilot trial, when the auriculotemporal nerve was blocked by infiltration over the posterior aspect of the zygoma anterior to the external auditory meatus and the greater auricular nerve, by infiltration of the cervical plexus at the central area of the posterior border of the sternomastoid muscle with bupivacaine 0.25%, there was a much lower incidence of, not only pain, but also vomiting after this operation. The similarity of these two reflexes, which either come from the musculature of the eye or the musculature of the ear to the emetic centre, requires more investigation to determine the real cause for these reflexes and how they are mediated.

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Effective peri-operative pain management in cancer patients

Patients with cancer often take large doses of oral controlled release morphine sulphate (OCRM, MST Continus). Delay in the absorption of these preparations in the postoperative period is recorded.[1,2] This has prompted the manufacturers to recommend that MST should not be used during the first 24 hours after surgery. The delay in gastric emptying which commonly occurs after anaesthesia and surgery might lead to accumulation in the stomach of MST given at regular intervals. The subsequent dumping from the stomach and absorption of this MST from the small intestine might result in dangerously high serum concentrations of morphine.[3] This risk is increased if initial delay in absorption postoperatively results in inadequate pain relief and further opioids are given parenterally.

Palliative surgery in cancer patients may be carried out for various conditions: fixation of pathological fractures or relief of intestinal obstruction for example. If the patient's pain control consists of regular doses of oral morphine, this needs to be replaced with, not only an equivalent analgesic parenterally peri-operatively, but an *increased dose* to cover the pain produced by the surgical intervention. We work as anaesthetists in a multidisciplinary Symptom Control/Support Team. Clinicians do not always correctly assess and appreciate the specific problems of patients on potent analgesics for chronic pain and we often notice the prescription of drugs which are inappropriate, such as buprenorphine or inadequate doses of drugs such as 50 mg intramuscular pethidine 4-hourly.

This group of patients needs to be recognised and realistic doses of potent analgesics should be prescribed.

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Epidural infusion of bupivacaine–fentanyl during labour

The recent paper by Reynolds and O'Sullivan (*Anaesthesia* 1989; **44:** 341–4) was interesting. They state Chestnut and colleagues also used rather large doses of bupivacaine with fentanyl by bolus and infusion, and found that the combination worked better than bupivacaine alone, though with no great advantage and in particular no improvement in normal delivery rate. They cite the published abstract[1] rather than the full-length paper,[2] which was published in May 1988. In neither the published abstract nor the full-length manuscript did we state or imply that the bupivacaine–fentanyl combination 'worked better than bupivacaine alone.' Rather, we concluded that 'infusion of the bupivacaine–fentanyl combination resulted in analgesia similar to that provided by infusion of a higher concentration of bupivacaine alone.'[1] We stated specifically, that 'the two groups were similar with regard to mean pain scores during the first stage of labour. ... Similarly, there was no significant difference between groups in patient assessment of analgesia quality during the first stage ... [or] the second stage. ...'[2] We did observe that women in the bupivacaine–fentanyl group had less intense motor block than women who received the higher concentration of bupivacaine alone. Reynolds and O'Sullivan correctly noted that women in the bupivacaine–fentanyl group did not have a higher incidence of spontaneous delivery.

The statement that our doses of bupivacaine and fentanyl were 'rather large', surprised me. In our full-length manuscript,[2] we reported that the mean (SD) doses of bupivacaine were 67 (32) mg in the bupivacaine–fentanyl group, and 99 (49) mg in the bupivacaine-only group. In five other published studies[3–7] of the continuous epidural infusion of bupivacaine alone, the mean (SD) doses of bupivacaine were 88 mg[3] 178 (83) mg,[4] 198 (63) mg,[5] 135 (59) mg,[6] and 161 (71) mg,[7] respectively. The mean dose of bupivacaine in the bupivacaine-only group in our study, exceeded the total bupivacaine dose of only one of these five studies. The mean (SD) dose of fentanyl in our study was 132 (23) μg.[2] The protocol was designed so that 'the maximal cumulative dosage of fentanyl was 150 μg.'[2] Reynolds and O'Sullivan did not report the mean total doses of bupivacaine and fentanyl in their study, but it is clear that some patients received at least 200 μg of fentanyl. Thus, we do not agree that our doses of bupivacaine or fentanyl were large.

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A reply

We are most grateful to Dr Chestnut for his interest in our article relating to the use of epidural fentanyl for perineal

pain in labour (*Anaesthesia,* 1989; **44:** 341–4). We are sorry we quoted from his abstract rather than the full length paper of 1988, but our original manuscript was prepared late in 1987, so we hope he will forgive us. We appreciate that Dr Chestnut and his colleagues did not *state* that the combination of bupivacaine with fentanyl worked better than bupivacaine alone. We were, however, misled by the figure in the abstract, which was not commented on in the text, but showed mean pain stores to be consistently lower with the combination, throughout the study period, and particularly between 60 and 150 minutes when standard error bars were very small indeed, and suggested that the majority of patients in the group who received bupivacaine and fentanyl were completely pain-free, while those in the other group were not. We were not sure of the power of their statistical methods to pick up this type of small but consistent difference. However, we must admit that in the full paper the figure shows the difference between the groups, though consistent, to be smaller, and we can well believe it was not significant.

We appreciate that the dose of bupivacaine used by Dr Chestnut and his colleagues was not large by comparison with many, and indeed the design of his study was superior to that of others in that he reduced the dose of bupivacaine when fentanyl was added. However, according to his protocol, patients in the bupivacaine–fentanyl group would have received 29 mg bupivacaine in the first hour and 37 mg in the first 2 hours, while their test dose alone contained 50% more bupivacaine than did our bolus dose.

Our study demonstrated that fentanyl 100 μg added to only 10 mg of bupivacaine in a bolus dose produced analgesia that lasted 140 minutes (*more* than 2 hours) in the course of an epidural block. Our earlier studies showed that 80 μg fentanyl together with 12 mg bupivacaine in a bolus at the start of epidural analgesia produced pain relief for a mean duration of 132 minutes.[1] So while we acknowledge that the dose of bupivacaine used by Dr Chestnut and his colleagues is not large taken out of context, it is one that might often be expected to be effective even without fentanyl, and it is large in comparison to our own. We do not deny that our dose of fentanyl was larger than his, but pharmacologically speaking these drugs are additive (probably *not* potentiating) and if more of one is used less is needed of the other. Our aim has always been to minimise local anaesthetic dose and side effects, though our achievement has only been rather more successful analgesia, particularly for perineal pain. We would emphasise the importance that pain is localised accurately in studies of obstetric analgesia.

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Reference

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Complete heart block

Drs Lonsdale and Stuart report on the development of complete heart block after the administration of glycopyrronium–neostigmine mixture to a hypertensive patient who received atenolol (*Anaesthesia* 1989; **44:** 448–9). They suggest that, in patients who receive beta adrenoceptor blocking agents, a higher dose of glycopyrronium than that incorporated in the commercially available mixture Robinul with Neostigmine (0.5 mg glycopyrronium with 2.5 mg neostigmine) should be used.

This is a good suggestion and furthermore we have shown[1] that excellent protection of the muscarinic effects of neostigmine can be provided by both simultaneous and prior administration of glycopyrronium with neostigmine. Glycopyrronium may be given initially in order to obtain an increase in heart rate before the administration of neostigmine in patients with pronounced bradycardia, or in those who receive beta blocking drugs.

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West Sussex RH10 2NH

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Reference

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Bradycardia after neostigmine in a patient receiving atenolol

The letter from Drs Lonsdale and Stuart (*Anaesthesia* 1989; **44:** 448–9) described a 45-year-old woman who received 100 mg atenolol once daily because of hypertension, and developed complete heart block with a ventricular rate of 35 beats/minute after reversal with glycopyrronium 0.5% mg with neostigmine 2.5 mg. They then injected 0.6 mg atropine and the heart rate changed to 90 beats/minute.

Our patient,[1] who showed an adverse atenolol–neostigmine interaction, did not respond to 1 mg atropine with 10 mg ephedrine sulphate. It is surprising how quickly the patient described by Drs Lonsdale and Stuart responded to only 0.6 mg atropine. The authors neither mention the duration of the bradycardia nor the blood pressure.

Another approach to treat such patients is not to give them neostigmine at all, but to ventilate their lungs after operation until the neuromuscular blockade wears off.

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Reference

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Hypoxia during outpatient dental anaesthesia

Allen and his colleagues (*Anaesthesia* 1989; **44:** 509–11) report that 13 dental patients out of 40 became hypoxic when given 33% oxygen, and seven out of 40 when given 50% oxygen.

A chi-square test showed no statistical significance, but I consider that the authors could have made it clearer that the result does not exclude the possibility that the incidence of hypoxia may be halved when 50% oxygen is used. Decreases of oxygen saturation of more than 10% are rare when 50% oxygen is used.[1] Nevertheless, I agree that it is also important to avoid airway obstruction during the surgical procedure.

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Reference

1. Parbrook GD. Training for day case dental anaesthesia. Oxygen saturation during general anaesthesia administered by dental undergraduates. *Anaesthesia* 1985; **40:** 377.

The paper by Allen, Rowbotham and Nimmo (*Anaesthesia* 1989; **44:** 509–11) was interesting since we have recently completed a study of 129 children for multiple extractions at a community dental clinic.

Their ages ranged from 2–15 years, and a similar anaesthetic technique to that described was used, with 2.5% halothane in 30% oxygen and 70% nitrous oxide. These dental chair anaesthetics were given by the same consultant anaesthetist throughout the study and all extractions performed by one of three experienced community dental officers.

Our results showed only seven children (5%) suffered episodes of hypoxaemia (less than 90%), and the range of these saturations was 83–89%. The equivalent group in Allen's study, Group 1, who received 33% inspired oxygen, had 13 children (32.5%) in the hypoxaemic category (range 29%–88%). Previous studies[1,2] examined oxygen saturation during dental anaesthesia and showed similar large decreases in saturation, but anaesthetics were given by junior anaesthetists and dental undergraduates, and extractions performed by junior or undergraduate dentists. We concluded that the experience of both the anaesthetist and the operator were major contributory factors in the reduction of the hypoxia in the dental chair.

A 32.5% incidence of hypoxaemia for dental anaesthesia is far from satisfactory, and the statement that there is no association between grade of surgeon or anaesthetist and the incidence of hypoxaemia must be viewed with caution. Perhaps one should stress the importance of experience in this specialty?

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References

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A reply

Thank you for the opportunity to reply to the comments raised by Drs Parbrook and Evans.

We agree with Dr Parbrook that the incidence of hypoxaemia was almost halved in the 50% oxygen group in our study. We are not surprised by this finding, but the fact remains that it was not statistically significant. Whether statistical significance would have been reached in a larger study is open to question. Wainwright *et al.* in a similar study of 100 patients published recently,[1] also failed to demonstrate any significance. However, we agree with Dr Parbrook in recommending that 50% oxygen should be used for dental outpatient anaesthesia.

We agree also with the opinions of Dr Evans, despite the fact that in our study we did not find a statistically significant correlation between hypoxaemia and grade of anaesthetist or surgeon. Dr Evans' data do not compare the incidence of hypoxaemia and the grade of anaesthetist or surgeon directly, but the low incidence of hypoxaemia is impressive. It is our opinion also that dental anaesthesia should be given only by experienced anaesthetists.

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Reference

1. WAINWRIGHT AP, PURDY G, FLYNN PJ. The use of high inspired oxygen concentration for dental outpatient anaesthesia. *Todays Anaesthetist* 1989; **4:** 4–6.

Cord examination after thyroidectomy

It was interesting to read the method of cord examination described by Dr V.J. Sarma (*Anaesthesia* 1989; **44:** 531).

We use a different method to view the function of vocal cords during operation. We use the fibreoptic nasolaryngoscope intra-operatively to identify the function of the recurrent laryngeal nerve in difficult cases.[1] The fibreoptic-laryngoscope is introduced after tracheal intubation into the larynx through the nose and the viewing end is positioned close to the anterior commissure. General anaesthesia is maintained without muscle paralysis. The identity and function of the recurrent laryngeal nerve is confirmed by stimulation of the suspected tissue with a disposable nerve stimulator (Pulse Atron catalogue No 62002) and the vocal cord movement observed through the fibreoptic laryngoscope. The fibreoptic cable is secured to the nasal vestibule with adhesive tape. The viewing end can be repositioned without difficulty if the patient's head moves during the procedure.

This method not only helps to identify the nerve, it also confirms the integrity of it at the end of the procedure; this may be of medicolegal importance.

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Reference

1. PREMACHANDRA DJ, RADCLIFFE G, STEARNS MP. A technique for intraoperative identification of recurrent laryngeal nerve and demonstration of its function. Accepted for publication. *Laryngoscope.*

Sticky labels to indicate hazard for anaesthesia

It is usually the practice, when an anaesthetist has a significant problem with a patient, to record it on the anaesthetic sheet.[1] This is often accompanied by a written warning on the outside of the patient's notes. Sometimes this has been formalised by the use of a stamp on which the details can be filled in,[2] or a warning card may be given to the patient.[3]

These methods are admirable so far as they go, but they are limited by different local practices. We propose to introduce an 'Anaesthetic Hazard' sticker which can be attached to the outside of the patient's notes and on which details of the incident can be written by the anaesthetist. This would be accompanied by a card that can be similarly filled in and given to the patient. Janssen Pharmaceuticals Limited has generously agreed to print the stickers and cards and to distribute them to anaesthetists in the United Kingdom. An example of the text of the sticker and card is shown in Figure 1.

The effectiveness of this system will depend on how much it is used by anaesthetists. It is hoped that it will be viewed as an investment in patient safety and that the use of the system will become widespread.

Any anaesthetist living and practising in the UK who does not receive the hazard stickers and cards should write to Dr E.W. Gasgoigne, Janssen Pharmaceuticals Limited, Grove, Wantage, Oxon OX12 0DQ.

The Royal Victoria Infirmary,
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R.J. BRAY
I.R. FLETCHER

ANAESTHETIC HAZARD

Hazard:

Patient: Hosp. No:

Anaesthetist:

Hospital:

Tel: Date:

Produced as a service to anaesthesia by
Janssen Pharmaceuticals Limited

Fig. 1

References

1. ISAAC PA. Difficult intubation-forewarned is forearmed. *Anaesthesia* 1986; **41:** 1158–9.
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3. FROST PM. Patients with problems. *Anaesthesia* 1987; **42:** 328.

Haemodynamic response to insertion of laryngeal mask

Further to the letter by R. M. Griffin (*Anaesthesia* 1989; **44:** 530), we have also noticed the lack of data about the haemodynamic response to the insertion of the Brain laryngeal mask (BLM). We have embarked on a study which compares the haemodynamic response to laryngoscopy and tracheal intubation with that during the insertion of a BLM. The results indicate that during insertion of the BLM there is little or no change in systolic blood pressure or pulse rate, unlike the 30–50 mmHg increase in systolic blood pressure associated with laryngoscopy and tracheal intubation.[1]

We hope to publish the results of the full study shortly.

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E.T.S. FORREST

Reference

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Cuff herniation

Two recent cases have re-emphasised the need to check the integrity of plastic, disposable tracheal tube cuffs before use.

One patient was to have a hysterectomy. Her trachea was intubated orally with a Mallinckrodt size 8 plastic Magill-type tube with a low pressure cuff; the cuff was inflated only sufficiently to ensure a seal. It was noticed almost immediately that there was reduced air entry and poor movement on the left side of the chest. The tube was progressively withdrawn without improvement, until finally a situation of almost complete airway obstruction developed with no air entry to either lung. The cuff could be seen on laryngoscopy to be just below the vocal cords. The cuff was deflated and ventilation to both lungs became normal. The tube was replaced and the case continued uneventfully. Examination of the tube showed a herniation of the cuff when inflated.

Orotracheal intubation was performed as part of a rapid sequence induction in a man who had a large bowel obstruction using a Mallinckrodt size 9.0 plastic Magill-type tube with a low pressure cuff. Ventilation was noted to be difficult with a high inflation pressure (4.0 kPa) and reduced expansion of the right side of the chest, although vesicular breath sounds were audible in all areas. Gradual withdrawal of the tube under direct laryngoscopy led to a sudden complete obstruction to ventilation. The cuff was deflated, whereupon ventilation became normal and air entry and movement became equal on both sides. The cuff was partially reinflated with a small volume of air and ventilation remained normal. Later examination of the tube showed a large herniation of the cuff when inflated.

It appears that in both these cases partial and finally complete airway obstruction occurred as a result of herniation of the tracheal tube cuff, which caused the end of the

tube to abut against the tracheal wall. The problem was resolved in both cases by deflation of the cuff. The herniation was not large enough in either case to protrude over the distal opening of the tube. Neither tube had a distal orifice (Murphy tip), the presence of which may have reduced the chance of this complication.

These two cases confirm the need to check the integrity of new, *plastic,* tracheal tube cuffs before use.

Crosshouse Hospital,
Kilmarnock,
Ayrshire

I. Davidson
S. Zimmer

A reply

Thank you for the opportunity to reply. Our Quality Assurance Department have examined the tubes in question and found that the areas surrounding the potential herniation were within blueprint specifications. All the cuffs of our tubes are fully inflated and tested before packaging, so it is very unlikely that Dr Zimmer would have identified any potential problem before tracheal intubation.

Examination of the bulge in the cuffs appears to indicate over-pressurising and stretching, which has been known to be associated with too deep intubation, coupled with increased pressure, either by overinflation or by nitrous oxide diffusion. We consider that this may have been exacerbated by withdrawal of the tube within the trachea whilst it was still inflated. Is this a common practice amongst anaesthetists? The tube may have been placed too far into the trachea.

Mallinckrodt (UK) Ltd,
Round Spinney,
Northampton NN3 4RQ

D.G.L. Wood

Is there liquid in the vaporizer?

A potentially serious problem has occurred on five separate occasions at three different hospitals with the Ohmeda Enfluratec Mark 4: a fluid level was visible in the sight glass when in fact the vaporizer is empty.

My first experience of this phenomenon was during a list of check cystoscopies. The second patient on the list was breathing enflurane in a mixture of nitrous oxide and oxygen, but continued to respond to attempts to insert the cystoscope despite an increase in the (enflurane) vaporizer dial to 5%. The sight glass on the Tec 4 vaporizer showed a 2-mm column of fluid. However, smelling the gases being emitted from the anaesthetic system suggested far less than 5% was actually being delivered. The patient quickly settled on halothane, and a different enfluratec was requested. When the faulty vaporizer was removed from the backbar it was tilted slightly, and the fluid level in the sight glass disappeared. Indeed, tilting the vaporizer completely on its side failed to demonstrate any fluid. On filling the vaporizer, it took rather longer than normal before fluid became visible in the sight glass, which suggested that the wicks had dried out. The vaporizer subsequently performed satisfactorily.

Some weeks later in another hospital, a patient ventilated with oxygen, nitrous oxide and 2% enflurane during an otherwise uneventful orthopaedic procedure began to show evidence of increased sympathetic activity with a progressive increase in blood pressure and heart rate. A review of proceedings revealed no obvious cause for this, but the fluid level in the Tec 4 sight glass was in a similar position to that described above. Removal of the vaporizer from the Selectatec manifold and a slight tilt caused the fluid level to disappear. On refilling the vaporizer and turning it back on to 2% the patient quickly settled again.

Further experience with a recent similar chain of events, I now watch the sight glass more carefully and have found two empty vaporizers that pretended to contain enflurane before the appearance of clinical signs to suggest lightening of anaesthesia.

The false fluid level appears to sit on a bubble which bursts when the vaporizer is fitted. Other anaesthetists have now reported the same phenomenon so it is difficult to believe that this only occurs in the hospitals at which I work.

The Operator's Manual for the Ohmeda Tec 4 vaporizers state that the vaporizer will function satisfactorily as long as there is agent visible in the sight glass. However, the manual also states repeatedly that the vaporizer should only be filled with the control dial in the OFF position. A number of my colleagues (none of whom have read the Operator's Manual) sometimes have difficulty filling the Tec 4 vaporizers, and resort to either filling with the vaporizer ON, or slightly unscrewing the filling adaptor. It may be that either of these manoeuvres introduce bubbles into the vaporizer chamber which leads to this false indication of anaesthetic agent in the sight glass.

The signs of inadequate anaesthesia were obvious in my first patient who was breathing spontaneously. However, if a patient were paralysed and his lungs ventilated it would be easy to misinterpret signs of increased sympathetic activity if the sight glass of the vaporizer indicated it contained anaesthetic agent: this situation could cause awareness.

Royal National
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Stanmore,
Middlesex HA7 4LP

J.P. Barcroft

A reply

We appreciate the opportunity to investigate the difficulty encountered by Dr Barcroft and to be able to respond.

We have not received any other reports of a similar nature. The company was immediately on the alert for similar events and, at the same time, we attempted to reproduce the observed effects by creating the conditions that we felt were most likely to have caused them. These attempts have been unsuccessful although, during the last 12 months, we observed something very much like this effect in two vaporizers during the manufacturing process. However no conclusion should be drawn from this because our processes differ from those normally carried out in a clinical environment.

The problem that Dr Barcroft has experienced will now be overcome by the new British Standard for 'Anaesthetic and analgesic machines', BS 4272: Part 3 3:1989. Ohmeda has actively participated, along with other UK Anaesthesia Machine manufacturers, in the production of this British Standard and clause 14.2, of the Standard, is particularly relevant. This is a requirement that the liquid level indicator fitted to 'Concentration-calibrated vaporizers' shall have a minimum level mark, as well as a maximum, and this feature is now on the Tec 4 vaporizers.

Details of this requirement are being incorporated into the Operation and Maintenance Manual for the Tec 4 range of vaporizers, and will require the user to refill the vaporizer before the minimum level is reached, thereby avoiding the difficulty experienced.

British Standards do not place an obligation on manufacturers to up-date all existing vaporizers but Ohmeda will introduce this feature, at no extra cost, on all Tec 4 vaporizers processed through our Service Centres.

We thank Dr Barcroft for bringing this matter to our attention thereby providing Ohmeda with an opportunity to improve one of our products.

Product Assurance Coordinator, B. SMITH
Ohmeda,
Steeton,
West Yorkshire BD20 2RB

Tracheal intubation with the patient prone

Cyst of the epiglottis reported recently by McHugh (*Anaesthesia* 1989; **44:** 522) and reviewed by others might be one of a number of situations in which tracheal intubation of the prone patient should be considered by the anaesthetist.

The technique is easy but rehearsal is essential so that the anaesthetist can give directions to the two or more assistants required should the need arise.

The patient is turned and lifted up the table with the head supported by one assistant so that the chin is clear of the top end of the table. This assistant is concerned solely with safeguarding the patient's head and neck and in lifting up the upper lip (particularly if there is a moustache) other assistants are required to position the patient.

Intubation follows the usual pattern but that the inverted laryngoscope is held in the *right* hand and the tracheal tube in the *left* with its curve concave towards the floor. The anaesthetist will find it easiest to kneel.

It is preferable to fix the tracheal tube into position after the team has completed repositioning if the proposed operative procedure is to be performed with the patient supine.

Quercus, H. MIDDLETON
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East Sussex TN33 9AA

Anaesthesia in myotonia dystrophica

The report by Drs Pollard and Young (*Anaesthesia* 1989; **44:** 699) in which they describe the use of propofol in a patient with myotonia dystrophica and found that there was a normal recovery at the end of surgery contrasts with my case in which the patient demonstrated prolonged somnolence after a propofol, nitrous oxide, oxygen, isoflurane anaesthetic on three occasions.[1]

It is interesting to speculate about the explanation for this increased sensitivity to propofol. My patient was allowed to breathe spontaneously but their patient received controlled ventilation of the lungs for a longer period. Carbon dioxide retention in spontaneously breathing patients is a possible cause of delayed recovery,[2] although there was no capnographic evidence of this. My patient may also have received albeit briefly a greater concentration of volatile agent during anaesthesia. It is possible that controlled ventilation of the lungs in these patients with a subsequently lighter plane of anaesthesia after a propofol induction is the answer.

University of Sheffield, K.A. MILLIGAN
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References

1. MILLIGAN KA. Propofol and dystrophia myotonica. *Anaesthesia* 1988; **43:** 513–4.
2. MILLER J, LEE C. Muscle diseases. In: KATZ J, BENUMOF J, KADIS LB, eds. *Anaesthesia and uncommon diseases: pathophysiological and clinical correlations.* Philadelphia, PA: W.B. Saunders, 1981: 533–7.

Anaesthesia in the Third World

A new edition of a text book is, as far as I know, the first which offers an analysis of dire problems which afflict the large part of humanity.[1] Professor J.L. Couper describes the issues, the challenges and the possible solutions to the problem of the provision of safe anaesthesia to all patients irrespective of social, cultural, economic, political or geographic boundaries.

Proper mention is made of the commendable educational projects of the World Federation of Societies of Anaesthesiologists (WFSA). These were considered in 1964 in a Panel Discussion on the 'Problems of Anaesthetics in Developing Regions' chaired by Professor Sir R.R. Macintosh during the 3rd World Congress of Anaesthesiology held in São Paulo. Proper emphasis is given to the role of simple and safe techniques of local infiltration and regional anaesthesia.

The first WFSA Training Center is in Caracas, Venezuela, (*South,* not Central, America) and it is to be hoped by the time of a future edition the impact of the WFSA's educational activities will be felt. These activities are receiving added impetus after the successful 9th World Congress of Anaesthesiologists held in Washington.

The lucid chapter draws attention to areas of human interest which are wider than anaesthesia itself. Such are problems of endemic and epidemic disease control, undernutrition, lack of or poor housing, water access, sewage disposal, educational and working opportunities and the ultimate lack of all, hope of any change in the foreseeable future. This alienation is potentially catastrophic. Proper hospital facilities, including anaesthesia, necessarily go *pari passu* with the fulfillment of those basic needs. The existence of adequate numbers of well trained and motivated physicians and ancillary personnel is a *sine qua non.* The more such disgraceful conditions are scrutinised the sooner they may be overcome.

The world already has sufficient capability to implement adequate solutions. However, cooperative efforts, based on common understanding, are essential to bridge the chasm between the euphemistically called developed and developing regions of our planet. Both, in fact, are contributing,

in diverse ways, to the paradox of the twentieth century: extremes of knowledge and of ignorance both thriving on an abundance of irrationality. Science and Technology should not be divorced from Philosophy and Ethics if we wish to face a brighter twenty-first and subsequent centuries. The alternative may be no future at all.

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C. PARSLOE

Reference

1. COUPER JL. Anaesthesia in the Third World. In: NUNN JF, UTTING JE, BROWN BR Jr., eds. *General anaesthesia*, 5th edn. London: Butterworths, 1989: 814–22.

A defect in a catheter mount

We wish to report a fault found in an Intersurgical catheter mount 2500. This consisted of a defect in the tubing at the 22 mm female connector end, proximal to the circuit connector port (Fig. 1). Less obviously this defect in the wall became a flange which had bent inwards to occlude the lumen of the tube. This obstruction was near total and prevented ventilation. The defect was spotted quickly and the catheter mount replaced.

The catheter mount and package were returned to the manufacturers who reported that they had no similar previous problems and have subsequently improved their quality control.

We suggest that an examination of the catheter mount lumen should be part of the preoperative equipment check, especially if the disposable type is being used.

Craigavan Hospital,
Co Armagh,
BT63 5QQ

C. MCALLISTER
I.A. ORR

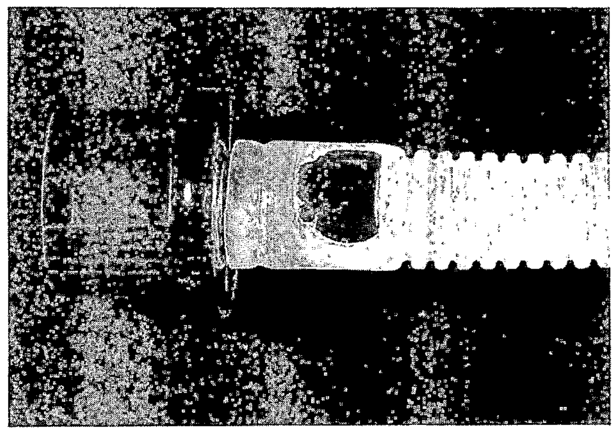

Fig. 1

Length of tracheal tubes

Correct tracheal tube placement is essential and differences in patient size and anatomy require that the tube's position be verified shortly after insertion. The greater duration and mobility of patients who receive intensive care often leads to radiological assessment of position and frequently necessitates trimming of the tracheal tube to an appropriate length. Observation of problems that occur when this is done with the aid of forceps to hold the tube to prevent its inward movement have led us to adopt this technique.

The desired length of the tube is determined either radiologically or by observation of the cuff as it passes through the vocal cords. The tube is cut at the desired length through only 75% of the circumference, the tube is then bent open at this level. The connector, previously loosened in its original site at the end, is inserted whilst the tube is stabilised by grasping the part of the tube that is in excess. This allows the insertion of the connector with safety, after which the remaining quarter of the circumference can be divided and the excess discarded.

This provides in our experience a rapid and secure means for insertion of the connector when tracheal tubes are shortened.

Whiston Hospital,
Merseyside L35 5DR

R.R. MACMILLAN
C.M. SAFFMAN

Correction

The letter from Dr M.K. Onsiong (*Anaesthesia* 1988; **43:** 907) should have been entitled 'Potential hazard of Hudson-Type Facemask'.

We have been asked to inform readers that the sole importers and distributors for Hudson Oxygen Therapy Sales Company (California, USA) are Henley Medical Supplies Limited. The mask to which reference was made is not manufactured by this Company but by Lifecare Hospital Supplies Limited (Market Harborough).

Anaesthesia, 1989, Volume 44, pages 942–944

Book reviews

Anaesthesia, Vols. 1 and 2

Edited by W.S NIMMO AND G. SMITH. Pp. xiii–1496. Blackwell, 1989. £95.

This book sets out to be the key to success in the Part III examination of the College of Anaesthetists. It contains all the information required by a candidate who already has a sound background knowledge of medicine and surgery. The scientific foundations are so clearly presented that the book will also serve the Part II candidate. Both teachers and taught now have a reference book that is up-to-date and well written.

The range of topics covered reflects the current breadth of anaesthetic subspecialties outside the operating theatre such as obstetrical analgesia services, intractable pain and the provision of intensive care. Current practice is also reflected in the increased emphasis given to regional anaesthetic techniques.

The text is presented in two tomes and is divided into five sections. Section I covers the Application of Scientific Principles to Clinical Practice. The physiology and pharmacology of sleep, the molecular basis for anaesthesia and the physiology and pharmacology of pain logically introduce the more specific chapters on physiology and pharmacology. Further chapters cover physics, gas supplies and flowmeters, ventilation of the lungs and fluid replacement therapy in the same detail. This section has 22 chapters and 429 pages.

Section II is entitled 'General Anaesthesia' and, after chapters on Operating Department and Recovery Room Design and on Morbidity and Mortality Studies, it describes the state of the art of general anaesthesia including most surgical specialties and specific problem patients such as the obese, the elderly and emergency or traumatised patient. There are also chapters on cardiopulmonary resuscitation and on AIDS and hepatitis B. The chapters vary in style from the diffuse and discursive to the dogmatic. Many authors have only enough space to summarise the main principles and problems in their field, indeed trauma is confined to six pages. The authors refer the reader to the appropriate monograph for more detailed study; this has often been written by the same author.

This reviewer accepts that a single chapter cannot replace a whole textbook on the same subject but hopes that future editions will expand this clinical part of the book.

The second volume begins with Section III on Local Anaesthesia and comprises eight well written chapters which are handicapped by barely adequate line drawings without the use of colour; grey on grey photographs are used to illustrate the techniques. The effect of this economy by the publishers is that many readers will choose to use alternative sources of information in this field.

Section IV covers pain; its assessment and management in acute and chronic situations, although the physiology and pharmacology of pain is given in Section I.

Section V is devoted to Intensive Care. It has an excellent introduction by Aitkenhead and is generally well written. It contains chapters on respiratory, renal and cardiac failure as well as burns, head injury and neurological disease. This format has led to the omission of specific topics such as chest injuries and pancreatitis.

Apart from the grey on grey photographs, the publishers have chosen black on grey for tables. This certainly does not produce clarity: perhaps it is meant to discourage photocopying. Nevertheless, the book is well produced, it has a large number of well chosen references and these are generally to 1987. The index is clear and highlights the main pages which deal with a particular topic.

The editors have assembled the foremost authorities in anaesthesia and intensive care and have succeeded in putting their views together in a readable, comprehensive and altogether outstanding textbook. Both British and overseas anaesthetists of all grades of experiences should buy it.

R. GREENBAUM

Safety and cost containment in anaesthesia

Edited by J.S. GRAVENSTEIN AND J.F. HOLZER. Pp. xiv + 257. Butterworth, 1988. £22.95.

The title of this book is not immediately appealing which is a pity because it contains much that will interest anaesthetists. It is the outcome of a workshop held at the University of Florida in 1987 at which 23 contributing speakers from various fields that included anaesthesia, law, insurance, health care management and manufacturing met to discuss safety and cost containment in anaesthesia. The result almost inevitably is somewhat of a curate's egg. The commonality between the speakers is one of balancing the cost of safety against profitability, and the provision of anaesthetic monitors against malpractice vulnerability. The emphasis on cost containment and financial decision making does not always make for comfortable reading.

The book explores the nature of the risk in anaesthesia, the cost of mishaps and how safety can be improved. It also explores less familiar areas of financial decision making, budgeting and the setting of minimal standards. The text epitomises the differences that exist between the UK and the US health care system, but it is a timely source of advice on the results of the separation of providers and purchasers of health care, setting up of contractual arrangements and the consequences of the equivalent of Crown Indemnity. The Resource Management Initiative and the White Paper make it necessary that we understand

such costing arrangements. There are excellent accounts on the nature of the risk in anaesthesia and the value of clinical outcome programmes which have only recently been established in this country. The problem of improving safety in anaesthesia is seen through the eyes of risk managers, practising anaesthetists, health care managers and lawyers. The latter explains with examples why doctors end up in court and this chapter should be read by all anaesthetists. Decisions on the purchase of monitors are made on the basis of profitability, risk reduction, and improved patient care. Will this be the pattern in this country in years to come?

The book ends on a more familiar note and discusses the setting up of minimum standards of monitoring. There is a realistic evaluation of the pros and cons of standardised monitoring and the use of automated records which are now entering the market. One is sympathetic to the fears of doctors that aggressive attorneys will use artifactual data and error corrections inappropriately in a court of law. Similar worries concern the recertification of anaesthetists and one wonders if this will be used both as a method of advertising one's competence and as a basis for a no-claims bonus on insurance premiums. Included in the book is a fascinating account of how pilots are trained using high fidelity simulators which pose no risk to passengers and it is suggested that this type of training could be extended to anaesthetists.

This is a wide ranging collection of articles from several disciplines concerned in the United States health care system and it would be difficult not to find something of interest for everyone. It is well written with a few clear and concise illustrations. I have enjoyed reading this book. I will no doubt refer to it again and recommend it to all who are interested in patient safety, the contract between patient and doctor and the proposed arrangements between providers and purchasers of health care.

R.W. Buckland

Textbook of pain, 2nd edn

Edited by P.D. Wall and R. Melzack. Pp. xiii + 1064. Churchill Livingstone, 1989. £95.00.

The new edition of this classic text on pain has been awaited with interest. It has undergone substantial revision and the list of contributors now includes the names of many famous and erudite people in the worlds of neurochemistry, neurophysiology and pain management. The introduction by Patrick Wall is an elegant treatise on pain, its contemporary definitions, causations, progression and remedies. He states the basic philosophy of the book. This is the premise that a unique specialised pain system (much like a burglar alarm system) does not exist but that pain is merely one of the options available for sensory experience.

The book is divided into three parts. The first addresses the basic concepts of pain, and groups these into peripheral, central and psychological aspects. Section two deals with clinical diseases in which pain predominates, while the third section attempts to provide some therapeutic answers. All topics are considered in depth and with extensive and generally up-to-date references. The illustrations are adequate and the index good.

What is the target audience of this large book? Because of its size and title it is unlikely to be read in depth by any others than those interested in the treatment of chronic pain. This is sad because it contains much of value to anaesthetists and to doctors in general. An excellent chapter on acute and postoperative pain by Michael Cousins should be read by all anaesthetic trainees, while the equally comprehensive chapter on labour pain by Bonica and Chadwick is an excellent introduction to the topic of obstetric analgesia. The chapters on the pain of burns, on the assessment of pain, and on its psychological elements all contain valuable information for anaesthetists. Those who have a special interest in chronic pain will find this book a comprehensive source of references to aid their understanding, diagnosis and treatment of pain problems. The methods of management described are in general up-to-date, although even here to quote Patrick Wall in the introduction, 'a few intellectual dodos honour the moribund corpse'!

Many of the chapters in section two would be of equal value to specialists in the relevant field: pain in the eye or cardiac pain rarely present first to the anaesthetist or even to the pain specialist.

If this book has a fault it is that of trying to be all things to all people. This is hardly surprising, since pain is one of the commonest presenting symptoms in medicine. All doctors would benefit from reading the excellent introduction. They would then understand when referral to a specialist in chronic pain would be indicated. They would also learn what not to do in an attempt to treat such problems themselves.

This book should, therefore, not only be in the possession of every specialist in pain relief, but also accessible to all anaesthetists and in every postgraduate library in the hope that other members of the medical profession may be tempted to browse!

D.E. Humphrey

Heart-lung interactions in health and disease

Edited by S.M. Scharf and S.S. Cassidy. Pp. xxv + 1135. M. Dekker, 1989. $175.00 (USA and Canda) $210.00 (all other countries).

This is no less than the 42nd volume in the prestigious series 'Lung Biology in Health and Disease' edited overall by Claude Lenfant. As might be expected from a series with this title, only a minority of the earlier volumes have covered cardiovascular function but here, in a little over a thousand pages, two American Professors of Medicine assemble contributions from more than 50 international experts to cover the lungs and circulation together.

The book is divided into three unequal parts — basic physiology (approximately 45%), pathophysiology (15%) and clinical applications (40%). Each in turn is subdivided into chapters, some grouped under specific subheadings. All are characterised by an intellectually demanding and rigorous analysis of current knowledge, emphasising where uncertainty and controversy lie and where new information needs to be sought. The academic anaesthetist and, above all, the academic intensive care clinician will find much to stimulate and inform, whereas those who seek a didactic practical manual must look elsewhere.

Could the information contained between these covers be found elsewhere? Could it be more up to date? Yes, for those willing to scour the literature, read and evaluate for themselves, there is little doubt that more could be found, published more recently. But as a comprehensive, well-referenced, well-illustrated and beautifully produced compendium of cardiorespiratory physiology, this book is hard to beat. It starts with oxygen transport and then examines how it is controlled in health, influenced by disease, investigated when abnormal and manipulated to achieve therapeutic benefit. Extreme circumstances, such as aerospace medicine and the physiology of cardiopulmonary resuscitation are included — in adjacent chapters, but this

apparent anomaly is no more than an index of the breadth with which the subject is covered. This is a book you will either covet or reject outright, depending on your response to the theoretical basis from which each topic is approached. What no-one can do is to deny its quality.

M. BRANTHWAITE

Textbook of paediatric anaesthetic practice

Edited by E. SUMNER AND D.J. HATCH. Pp. viii + 603. Baillière Tindall, 1989. £37.50.

This book fulfils the need for a moderately-sized yet comprehensive textbook of paediatric anaesthesia. The editors, both experienced authors, have been fortunate in persuading so many other experts to contribute to what must surely become an important contribution to the literature.

True to the book's title, most of the 23 chapters are concerned with practical aspects of paediatric anaesthesia, although there are also excellent chapters on the pharmacology of anaesthetic agents, muscle relaxants and fluid and electrolyte balance. All the chapters are well-written, adequately illustrated and amply referenced. Perhaps the only exception to this is the chapter on basic techniques of paediatric anaesthesia which lacks a proper introduction.

The following deserve special mention. Dr Feychting's chapter on psychological preparation for anaesthesia, which emphasises the importance of the personal visit of the anaesthetist, and the support which can be provided by parents at induction of anaesthesia. Dr Battersby brings considerable knowledge and a wealth of clinical experience to his chapter on monitoring. His uncompromising position on minimum monitoring equipment is welcomed. Dr Armitage's chapter on local anaesthesia is a model for less experienced authors. The role of regional blocks in paediatrics is clearly defined, individual blocks are adequately described and the author's preference is stated. Dr James tackles the difficult subject of anaesthesia for emergency surgery with enthusiasm, emphasising the need to restore circulating volume before anaesthesia and prevent regurgitation of gastric contents at induction. Finally, Dr Keneally offers excellent advice on the selection and management of children undergoing day-stay surgery.

In general, the editors have chosen their chapters wisely. The inclusion of chapters on upper airway obstruction and resuscitation in paediatrics seems entirely appropriate, as does the chapter on congenital anomalies, which provides useful reference material and a number of excellent photographs. However, I would question the inclusion of separate chapters on anaesthesia for liver and heart transplantation, and a chapter about medical facilities in the Third World. These chapters whilst topical, deal with few problems specific to children and one wonders whether the general anaesthetist, for whom the book is primarily intended, would have gained more from a chapter on paediatric intensive care.

In summary, I thoroughly enjoyed reading the first edition of this important new book. Its purchase is recommended to all with an interest in paediatric anaesthesia.

G. MEAKIN

Books received

We thank the publishers for the following books, some of which may be reviewed in future issues of the journal.

Cardiopulmonary bypass: current concepts and controversies
Edited by J.H. TINKER. Pp. x + 156. W.B. Saunders, 1989. £30.00.

Talking health
Edited by SIR JAMES WATT AND C. WOOD. Pp. 167. Royal Society of Medicine, 1988. £4.95.

Clinical problems in acute care medicine
Edited by J.J. HEFFERNAN, R.A. WITZBURG, A.S. COHEN. Pp. xxvi + 731. W.B. Saunders, 1989. £25.50.

Capnography in clinical practice
J.S. GRAVENSTEIN, D.A. PAULUS, T.J. HAYES. Pp. x + 158. Butterworth, 1989. £21.95.

Transfusion medicine
Edited by W.H. Churchill. Pp. xii + 366. Blackwell, 1989.

Case presentations in respiratory medicine
J.A. ELLIOTT. Pp. x + 211. Butterworth, 1989. £11.95.

Anesthesia and the heart patient
Edited by F.G. ESTAFANOUS. Pp. xiii + 367. Butterworth, 1988. £50.00.

Medicine for anaesthetists, 3rd edn
Edited by M.D. VICKERS AND R.M. JONES. Pp. vii + 600. Blackwell, 1989. £57.50.

Manual of intensive care medicine
Edited by J.M. RIPPE. Pp. xiii + 600. Churchill Livingstone, 1989. £9.95.

Clinical and resuscitative data, 4th edn
R.P.H. DUNNILL AND M.P. COLVIN. Pp. viii + 248. Blackwell, 1989. £16.95.

Essentials of critical care medicine
M.J. TOBIN. Pp. xiii + 578. Churchill Livingstone, 1989. £19.95.

Anaesthesia, 1989, Volume 44, pages 945–949

Anaesthetic literature

This section of *Anaesthetic literature* contains references taken from *Current Contents—Life Sciences* for July 1989. The Journals which may be consulted when *Anaesthetic literature* is compiled are those listed in *Current Contents—Life Sciences*, published by the Institute of Scientific Information, Philadelphia, Pennsylvania, USA.

Pharmacology

Adrenergic drugs and their antagonists

Pharmacodynamic and pharmacokinetic study of propranolol in patients with cirrhosis and portal hypertension. CALES P, GRASSET D *et al. British Journal of Clinical Pharmacology* 1989; **27:** 763.

Labetalol: a reappraisal of its pharmacology, pharmacokinetics and therapeutic use in hypertension and ischaemic heart disease. GOA KL, BENFIELD P, SORKIN EM. *Drugs* 1989; **37:** 583.

Catecholaminergic and opioidergic mechanisms involved in the hypotensive response of pindolol. JONES LF, TACKET RL. *European Journal of Pharmacology* 1989; **165:** 123.

Lipid effects of celiprolol, a new cardioselective beta-blocker, versus propranolol. SIRTORI CR, JOHNSON B *et al. Clinical Pharmacology and Therapeutics* 1989; **45:** 617.

Paradoxical effects of endothelin on cardiovascular noradrenergic neurotransmission. WONG-DUSTING HK, REID JJ, RAND MJ. *Clinical and Experimental Pharmacology and Physiology* 1989; **16:** 229.

Anaesthetic agents

Pharmacokinetics of the newer benzodiazepines. GARZONE PD, KROBOTH PD. *Clinical Pharmacokinetics* 1989; **16:** 337.

Atropine, diazepam and physostigmine: thermoregulatory effects in the heat-stressed rat. MATTHEW CB, HUBBARD RW, FRANCESCONI R. *Life Sciences* 1989; **44:** 1921.

Anesthetic effects on liver and muscle glycogen concentrations: rest and postexercise. MUSCH TI, WARFEL BS *et al. Journal of Applied Physiology* 1989; **66:** 2895.

Radioreceptor assay of clonazepam and diazepam in blood for therapeutic drug monitoring. NISHIKAWA T, NISHIDA A *et al. Therapeutic Drug Monitoring* 1989; **11:** 483.

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The collator of this section is Dr L. Kaufman, MD, FFARCS, 145 Harley Street, London W1N 2DE. Dr Kaufman is prepared to provide on request, and at a modest charge, a new additional service to our readers. References from January 1984 have been entered on data base. The data are held on Dbase II, cpm 86 and on Dbase II, MsDos and are available on disc, together with a program providing search facilities. Enquiries direct to Dr Kaufman at the above address please.

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Anaesthesia, 1989, Volume 44, pages 951–952

Editorial

Professor Emeritus Sir Robert Reynolds Macintosh MA, DM, FRCSE, DA, DSc(Hon)Wales, FFARCS(Hon), FFARACS(Hon), FFARCSI(Hon) FRCOG(Hon)
17 October 1897–28 August 1989

'He is greatest who is most often in men's good thoughts'. (*Samuel Butler*)

The death of Sir Robert Macintosh brings to an end a distinct era of anaesthesia. His life spanned the period during which anaesthesia changed from an activity thought hardly needing a doctor, to a distinct and valued branch of medicine. Macintosh played a major part in bringing about these changes.

A New Zealander by birth, Macintosh was a fighter pilot in the first World War. He took up medicine after the war, qualifying from Guy's in 1924. Originally intending to be a surgeon, he finally chose anaesthesia as his life work, and settled down in London where he established a highly successful private anaesthetic group practice.

The story of how the Oxford Chair in Anaesthetics came to be established in 1937, thus giving Anaesthesia the status of a University discipline, has already been told many times. Suffice it here to remember that at the time, anaesthesia was considered to be a lowly form of medical work. Indeed most surgeons were perfectly happy for anaesthesia to be administered by one of their junior assistants or by untrained General Practitioners. In most other countries, it was left to nurses and even hospital porters. The suggestion that the practice needed a medical training and should be considered a branch of medicine was fiercely resisted. In any case there was nowhere to be found any organised training in anaesthesia, probably because no one considered there was much to be learnt. The idea therefore of a Chair of Anaesthetics in the prestigious University of Oxford appeared as both laughable and outrageous. Fortunately Macintosh was friendly with Lord Nuffield, and was able to convince him that anaesthesia was an important part of medicine and that many aspects of its practice at the time were deplorable. When Nuffield endowed a number of medical academic departments in Oxford University, he insisted that anaesthesia should be included. So convinced was he that he made his lavish donations to Oxford *conditional* on anaesthesia being one of the departments.

The appointment of Macintosh as the first Nuffield Professor in 1937 surprised many because he had no obvious qualifications for the job. Nor indeed did anyone else. As it turned out the choice was an inspired one. This modest man, who throughout his life emphasised that he had no pretensions to being an 'academic', nevertheless made the Oxford department pre-eminent in the world of anaesthesia, with an impact on medicine far beyond the field of anaesthesia itself.

There was no precedent for a University Department of Anaesthetics in Britain, and therefore no model on which to build. Apart from looking at one or two such departments in the USA, visits which were on the whole of somewhat limited value since the socio-political background in that country was so different from our own, Macintosh had to develop his own department from scratch. He had the wisdom to surround himself with a small team of enthusiastic experts in such fields as physics, physiology, and pharmacology. Both he and they were fully aware of the limitations in the knowledge and practice of anaesthesia which was current at the time. The policy which governed every aspect of the new department was not just to enable anaesthesia to extend the limits of surgery, important though that was, but to make anaesthesia more safe.

Macintosh was first and foremost a clinical anaesthetist. Throughout his career at Oxford he was to be found in the operating theatres every day and often at night too. There, he was able to identify the shortcomings in anaesthetic practice and theory, and to set in motion research to rectify or improve matters. By his devotion to the clinical art as well as to the science of anaesthesia he set an example to many of the 'professors' in other countries, who were more likely to be found in an office or laboratory than in an operating theatre or hospital ward.

His contributions to practical anaesthesia were enormous. They may not have included much at the growing points of the biological sciences, but because they were largely concerned with everyday practical problems had a great impact on the safety and the flexibility of anaesthesia and thus on the progress of surgery. He wrote about and influenced almost every aspect of clinical anaesthesia, introducing new methods and instruments, most of which changed current practice in some way. His books and papers were models of clarity. As his First Assistant for many years, and a co-author of many books and papers with him, such abilities as I may have in writing, I owe largely to the carefully crafted, clear, and accurate style of medical writing he insisted on.

Macintosh was not an orator and he was not always comfortable when lecturing or reading papers. He loved teaching, and was at his best with a small group surrounding him in the anaesthetic room or operating theatre. To be present on such occasions was a delight and a privilege. Many a surgeon would pause while operating in order to listen to and understand his disarmingly simple explanation of some observed reaction on the part of the patient.

His appointment as Advisor in Anaesthetics to the R.A.F. enabled him to spread his practical approach to clinical anaesthesia emphasising safety and simplicity, to

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almost every British anaesthetist, with results still evident today. After the War his fame spread internationally and he was invited to visit almost every country which included anaesthesia in its medical vocabulary. He therefore had a great influence in shaping world anaesthesia.

He gave generously to the juniors and associates in his department. They were offered opportunities of all sorts; collaborating in or initiating research, attending scientific meetings, and writing papers. When they moved on, the help he gave them in their future careers was always unstinted. As a man, his virtues were many. His modest nature abhorred camouflage, dissimulation or evasion. He was always truthful in matters medical, particularly when admitting his own ignorance. He was never offensive to others and rarely upset anyone who disagreed with him. He got on well with surgeons, but there were occasional exceptions in the early days. He took the view that it was part of his job both to learn as much as possible himself, and to teach what he knew to others. This brought him into occasional conflict with surgeons who could not understand this. On such occasions he invariably gave a mild, disarming, and quiet response to any aggressive, rude or offensive remarks about anaesthetists in general and academic ones in particular, that came from impatient surgeons who had neither the wit nor the capability to understand the responsibilities and difficulties of teaching anaesthetics in a University Hospital.

Lord Nuffield was generous with his benefaction to Oxford University. The reluctant University, forced to include anaesthesia among the new departments, was less than generous to Macintosh and his department. He had a constant and long running struggle with the university authorities over such things as status, grants, and accommodation. The undercurrent of opposition to anaesthesia continued to dog him to the day of his retirement, and was carried over to his successors. While 17 other British Universities established Chairs in Anaesthetics, the University of Oxford made more than one attempt to abolish its own Chair of Anaesthetics. In spite of these difficulties, little known to his colleagues at the time, Macintosh remained imperturbable. In the event, the Medical Faculty of Oxford University became as much known internationally for its contributions to anaesthesia as for those in any other branch of medicine.

His knighthood in 1955 was acclaimed by all, and was evidence of recognition of his major impact on anaesthesia. His work was recognised abroad by Honorary Degrees from Universities in Argentina, France, and Poland. Regrettably this was not mirrored here. The only University in Britain to honour him and his specialty in this way was the University of Wales.

The death of his first wife Marjorie left him desolate. He was indeed fortunate to marry again to Anne whose devotion and support was obvious to all, and to whom Macintosh attributed the happiness of his retirement years.

Future historians of anaesthesia will see Macintosh as a great steadying influence on the development of anaesthesia. Although he took little part in the Institutions of anaesthetists, he gave his advice and support to them readily. He kept the craft of anaesthesia on an even keel, balancing it between the fundamentalists who would have anaesthesia stay with chloroform and the open mask, and the new breed of scientific anaesthetist who saw the future of anaesthesia to depend on the laboratory rather than on personal attention to the patient. We as anaesthetists, and the general public as well, should be grateful that Macintosh was amongst us for so many years. His passing brings to an end a most fruitful era in the development of anaesthesia.

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W.W. MUSHIN

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Anaesthesia, 1989, Volume 44, pages 953–958

Admissions to the intensive care unit after complications of anaesthetic techniques over 10 years

1. The first 5 years

A. L. COOPER, J. M. LEIGH AND I. C. TRING

Summary

Fifty-three patients were admitted in a 5-year period to the intensive care unit as a result of a complication of an anaesthetic technique. These patients represented 1 in 1543 anaesthetics carried out in the District in the period and 2.0% of all admissions to the intensive care unit. The mortality rate was 17%. The complication was considered to be wholly or partially avoidable in 14 instances (26%). Five of these subjects died and two had a residual neurological deficit.

Key words

Anaesthesia; audit.
Intensive care.

The practice of anaesthesia is beset with potential disasters. The fact that significant morbidity and mortality does not occur is a tribute to the high degree of skill acquired during the training of the modern anaesthetist. Nevertheless, morbidity and mortality do occur as a result of anaesthetic techniques and are highly significant to those unfortunate patients who suffer as a result.

It is easy to recognise anaesthetic mortality, but morbidity is more difficult to study. However, we have collected prospectively all the patients admitted to the intensive care unit (ICU) in our District whose main admission diagnosis was encoded as 'Anaesthetic Complication'. The purpose was to establish the range and incidence of different complications, to identify predisposing factors if any, and to comment on the avoidance of such incidents.

The 5 years reported here represent the first half of a 10-year prospective study period. The reason for the subdivision is that the rate of patients who have received surgical or medical procedures in the District in the 10th year is double that in the first year, while the number of staff in anaesthetics has not increased. Comparisons between the two periods will also be informative.

Methods

Details of all patients admitted to the intensive care unit at the Royal Surrey County Hospital from 1 January 1979 to 31 December 1983 with a main admission diagnosis of 'Anaesthetic Complication' were collected prospectively. The cases were those in which admission to the ICU was not planned before the operation. We have defined a complication of anaesthesia as 'an undesirable effect attributable to a practical procedure or drug used for analgesia or sedation, or as part of an anaesthetic technique, although not necessarily involving an anaesthetist'.

The hospitals in South West Surrey District provide services for all specialties except cardiac surgery and neurosurgery. The operating registers of all hospitals were collated so that the numbers and different types of anaesthesia or analgesia techniques carried out in the 5-year period were determined.

Results

Seventy-one thousand procedures were carried out in the 5-year period, under general anaesthesia; 5270 under intravenous sedation, with (or without) local anaesthesia; 2410 were with local anaesthesia alone and there were 3100 obstetric epidurals (total, 81 780). There were 2651 admissions to the ICU in this time and 53 were encoded for our computer records with a main admission diagnosis of 'Anaesthetic Complication' (2% of ICU admissions). The overall rate of complications which necessitated admission to ICU was therefore 53/81 780 (1 in 1543).

Time of identification of complication

Figure 1 shows that the majority (33 of 53) of complications occurred in the recovery period. The great majority of these were due to ventilatory inadequacy after reversal of

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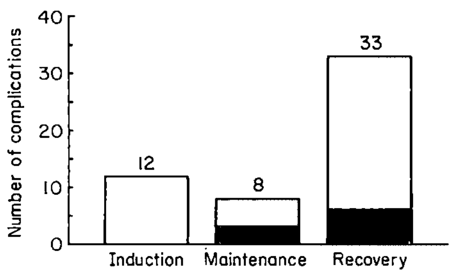

Fig. 1. Time of occurrence of complications in relation to anaesthetic sequence. Shaded area equals deaths; three during maintenance, six during recovery.

muscle relaxants. The causes of complications that occurred during induction, maintenance and recovery are summarised in Tables 1–3. No complications occurred after local or regional analgesia.

Sex and age

There were 23 men with an average age of 57 (SD 23, range 1–90) years and 30 women, average age 63 (SD 10, range 23–93) years. The entire group had an average age of 60 (SD 20) years.

Length of stay in ICU

The range of stay varied from 1 to 990 hours, but the patients could be divided into two subgroups on the basis of duration of stay. Forty-four patients each spent less than 75 hours in ICU, and occupied 22% of the total time (938 hours: mean time in ICU 21, SD 15 hours; SEM 2 hours; range 1–72 hours). The remaining nine patients, who spent more than 75 hours each, occupied 78% of the total time (3251 hours—mean time in ICU 361, SD 261; SEM 72 hours; range 103–990 hours).

The mean age of the patients who stayed less than 75 hours in ICU was 58, SD 22 years and the mean age of the patients who remained in the Unit for more than 75 hours was 67, SD 33 years. The death rate in the group who stayed less than 75 hours was 4/44 (9%) and the death rate in the other group was 5/9 (56%).

Emergencies

Twenty-seven patients were admitted after emergency operations and 26 after elective procedures. Ten thousand and eighty emergency operations were performed during the 5 years. Thus the risk of requiring ICU admission was 1 in 388 for emergency, and 1 in 2655 for elective cases; this is a seven-fold difference.

Table 1. Causes of complications during induction.

	Number	Deaths	Permanent morbidity	Avoidable factors present
Difficult or failed intubation	3	0	1	3
Aspiration	3	0	0	1
Severe bronchospasm	1	0	0	0
Suxamethonium apnoea	2	0	0	0
Hyperthermia	2	0	0	0
Hypersensitivity reactions	1	0	0	0
Totals	12	0	1	4

Table 2. Causes of complications during maintenance.

	Number	Deaths	Permanent morbidity	Avoidable factors present
Cardiac arrhythmia or arrest	7	3	1	4
Bronchospasm	1	0	0	0
Totals	8	3	1	4

Deaths

Eight patients died in the ICU and one within 24 hours of leaving it; this is an overall mortality rate within the group of 17%. Five of these were considered to be partially or totally attributable to the anaesthetic complication, and in the other four who died the primary pathology and surgical factors contributed mainly to their deaths.

Figure 1 demonstrates the relative risk of death between complications that occurred during maintenance or in the recovery period (three deaths in eight cases during maintenance (37.0%); six deaths in 33 cases in recovery (18%)).

Permanent morbidity

There were two cases of permanent morbidity. The sequence of events which led to the permanent deficits, which occurred as a result of hypoxic cardiac arrest, contained avoidable elements and have cost £0.6 million in damages and fees in uncontested legal hearings. No pulse oximeters were available in the District.

Complications which were wholly or partially avoidable

Fourteen cases in this category are discussed below.

Difficult or failed tracheal intubation during induction (Table 1)

The first of these patients was a young female with a narrow underslung jaw who was for Caesarean section. She already had an epidural block and during the induction of general anaesthesia, intubation was found to be difficult. Oxygenation by mask was not easy and, although cardiac arrest did not occur, anoxic bronchospasm supervened before the airway was established. Recovery was

Table 3. Causes of complications during recovery.

	Number	Deaths	Permanent morbidity	Avoidable factors present
Ventilatory failure	24	5	0	2
Aspiration	5	1	0	2
Respiratory obstruction	2	0	0	1
Acute pulmonary oedema	1	0	0	0
Hydrothorax	1	0	0	1
Totals	33	6	0	6

uneventful. It seems very difficult to justify the induction of general anaesthesia in this patient since analgesia was already adequate. The anaesthetist was a senior registrar.

The second patient might also have benefited from the provision of a spinal or epidural rather than a general anaesthetic. He was a very obese and asthmatic 68-year-old man with a hip fracture. He had a receding mandible and crowned front teeth. Intubation proved difficult and was accomplished blind with an introducer but not before regurgitation and aspiration had occurred. Recovery was complete after 16 hours in the ICU.

The final patient in this category was a male for a hip replacement. The registrar anaesthetist found intubation difficult and apparently failed to maintain oxygenation. Cardiac arrest occurred and, although intubation and resuscitation was successfully achieved by a consultant who was called from another theatre, the patient had permanent neurological sequelae.

Regurgitation and aspiration during induction

A patient who was known to have a hiatus hernia and was diabetic and who had had many anaesthetics for lower limb surgery was anaesthetised by a senior registrar. All previous procedures were carried out under either spinal analgesia or general anaesthesia with intubation. This was the only anaesthetic carried out by mask and he regurgitated and aspirated. Recovery was, however, without sequelae.

Cardiac arrhythmias or arrest during maintenance (Table 2)

The first of these cases was a 73-year-old woman for hip replacement who received induced hypotension with a ganglion blocker and suffered cardiac arrest after severe hypotension. The anaesthetist was a consultant. This operation is carried out by many anaesthetists without the use of induced hypotension.

The second patient in this category was undergoing middle ear surgery and again suffered a cardiac arrest during induced hypotension. He was known to suffer from vertebrobasilar insufficiency and it was probably unwise to use this particular technique in his case. The anaesthetist was a consultant.

Both these patients died within a short time of admission to the ICU.

A further case in this category occurred when cardiac arrest followed what was probably progressive hypoxia, as a result of either a kinked or displaced tracheal tube. The anaesthetist was a consultant.

The final case was one in which a trainee surgeon was carrying out endoscopy in a bleeding patient to whom he gave a relative overdose of diazepam. The patient became apnoeic and cardiac arrest occurred. The patient died.

Ventilatory failure during recovery after reversal of neuromuscular blockade (Table 3)

An elderly women with dystrophia myotonica required a cataract operation. The consultant anaesthetist chose general anaesthesia and intermittent positive pressure ventilation of the lungs (IPPV) via a tracheal tube. Quite soon after operation the patient developed respiratory failure, principally associated with sputum retention and she required a very long period of intensive care before she recovered. It is probable that these problems would not have occurred if this procedure had been carried out under a local block.

A 71-year-old man with a bowel obstruction regurgitated and aspirated during rapid sequence induction of anaesthesia for laparotomy. Neuromuscular blockade was reversed and extubation occurred at the end of surgery but his breathing was inadequate by the time he reached the recovery ward and he was admitted to ICU for mechanical ventilation but died a few days later. It is possible that this patient might have survived if elective rather than therapeutic ventilation had been used. The anaesthetist was a registrar.

Regurgitation and aspiration during recovery

Anaesthetists were not directly involved in either of these cases. The first was a 90-year-old man who vomited, aspirated and had a respiratory arrest 3 hours after his return to a surgical ward. The prescription of 15 mg papaveretum was clearly too generous. He made a complete recovery after a period of IPPV in the ICU.

The second of these cases was an elderly man with ischaemic heart disease who ultimately died of adult respiratory distress syndrome. He had regurgitated and aspirated while he was supine in a recovery ward after laparotomy for intestinal obstruction. This should not have happened but he had apparently turned himself over onto his back before he became more deeply unconscious.

Respiratory obstruction during recovery

This incident also occurred in a recovery ward where the patient had a cardiac arrest as a result of airway obstruction. He was successfully resuscitated without sequelae. It is unlikely that resuscitation would have been so prompt and successful had this occurred in any other environment.

Hydrothorax

Hydrothorax developed after the insertion of a central venous pressure (CVP) line. There were no sequelae to this event other than the temporary inconvenience of a chest drain. This CVP line was inserted from the antecubital fossa using a drum cartridge catheter into which there was initial bleed-back. However, after full insertion a further bleed-back test was not carried out and a litre of compound sodium lactate solution with a muscle relaxant was deposited in the pleural cavity. Perforation of one of the great veins is not common when a catheter is inserted via this route, but we consider that the deposition of the infusion solution in the pleural cavity might have been avoided. The anaesthetist was a consultant.

Grades of staff

Fourteen cases were considered to have avoidable components. Three were incidents in the recovery or general ward and one was at the hands of a trainee surgeon; so there were 10 cases in which the responsibility was that of anaesthetists. Five of these were anaesthetised by consultants, three by senior registrars and two by registrars.

Complications which were unavoidable or unpredictable

Regurgitation and aspiration during induction of mask anaesthesia

A female patient regurgitated and aspirated during induction of anaesthesia for elective surgery and, on investigation afterwards, was shown to have a hiatus hernia. The other patient in this group was properly starved before manipulation of a wrist fracture under anaesthesia and had no gastrointestinal pathology.

Severe bronchospasm during induction

Severe bronchospasm was observed in an obese, bronchitic smoker after a thiopentone, suxamethonium, tracheal intubation, fentanyl, pancuronium sequence. He was given aminophylline and hydrocortisone and discharged from ICU after 7 hours.

Suxamethonium apnoea during induction

Two patients who had prolonged apnoea after suxamethonium were admitted to ICU for ventilation. Both had abnormal cholinesterase activity and were counselled accordingly.

Hyperthermia during induction

Hyperthermia occurred in two patients. One was a 41-year-old male given thiopentone, suxamethonium and halothane for a middle ear operation. Dramatic improvement occurred with dantrolene and the diagnosis of malignant hyperpyrexia was subsequently confirmed.

The second case was a 23-year-old woman given thiopentone, suxamethonium and halothane for elective surgery. She was successfully treated with dantrolene and methylprednisolone. Subsequent investigation showed that she had myotonia congenita.

Hypersensitivity reactions during induction

A hypersensitivity reaction on induction occurred in one patient. A woman with arterial disease developed profound hypotension and bradycardia after thiopentone and tubocurarine given for an intended lumbar aortogram. She had excessive flare and wheal at the site of injection and subsequent tests demonstrated that she was sensitive to thiopentone. Resuscitation was successful.

Cardiac arrhythmias or arrest during maintenance

A 67-year-old male with a past history of hypertension, myocardial infarction and two minor cerebrovascular accidents had cardiac arrest for 30 seconds at the beginning of an Austin Moore hip replacement. Anaesthesia consisted of droperidol, fentanyl, thiopentone, tubocurarine, oxygen and nitrous oxide and he woke up soon after reversal with no ECG, cardiac enzyme or neurological changes.

A 66-year-old male went into bigeminal rhythm followed by 90 seconds of asystole during the application of cocaine paste to the nasal mucosa prior to septoplasty. Spontaneous recovery of the heart beat occurred after cardiac massage and there were no sequelae.

An 80-year-old diabetic developed multifocal ventricular ectopics during anaesthesia. Spontaneous recovery of normal rhythm occurred shortly after entry to ICU.

Bronchospasm during maintenance

A 26-year-old male with liver disease and portal hypertension developed bronchospasm during portal angiography under general anaesthetic. He recovered after 3 hours in ICU.

Ventilatory failure after attempted reversal of competitive (non-depolarising) muscle relaxants during recovery

This was the largest single group of admissions (24 of 53). Twenty-two of the 24 seemed to be unavoidable, although perhaps partially predictable. The most distinguishing feature of this subgroup was the average age (70, SD 12, years) compared with the 29 residual patients (average age 52, SD 22, years). Eleven of these were elective cases and 13 were emergencies. The majority (11 of 13) of the emergencies had laparotomies (700 performed during the study period). The elective cases represent a wide range of procedures (Table 4). The pre-operative clinical features of the 24 patients with postoperative ventilatory failure are summarised in Table 4 and the operative procedures carried out on them are summarised in Table 5.

Regurgitation and aspiration during recovery

Two elderly patients aspirated stomach contents in the recovery ward after laparotomy for intestinal obstruction; they were lying on their side at the time. Both required mechanical ventilation of the lungs in ICU and survived.

A 23-year-old woman aspirated after excision of a pilonidal sinus. She was clinically cyanosed and was admitted to ICU for observation; she recovered quickly.

Respiratory obstruction or lack of airway control in recovery

One patient with delayed recovery of consciousness and prolonged lack of airway control was admitted to ICU for observation. The patient recovered slowly without any obvious reasons other than possible sensitivity or relative overdose.

Table 4. Pre-operative clinical problems of 24 patients with postoperative ventilatory failure.

Clinical problems	Number of patients
Cardiovascular disease	8
Respiratory disease	6
Bowel obstruction/strangulation	7
Haemorrhage	3
Septicaemia	3
Carcinomatosis	3
Obesity	2
Dystrophia myotonica	1
Pneumothorax	1
None of the above	7
One of the above	5
Two of the above	9
Three of the above	3
More than three of the above	0

Table 5. Surgical procedures undergone by 24 patients with postoperative ventilatory failure.

Procedure	Number of patients
Emergency	
Laparotomy	11
Re-suturing abdomen	1
Abscess in groin	1
Elective	
Laparotomy	1
Cholecystectomy	2
Hysterectomy	1
Sterilisation (open)	1
Mediastinoscopy	2
Heller's procedure	1
Cataract extraction	2
Arteriography	1

Acute pulmonary oedema

An 18-year-old coloured patient with sickle cell trait required a facial laceration to be sutured. Anaesthesia consisted of nitrous oxide, oxygen and halothane, with spontaneous breathing via a tracheal tube. He stopped breathing on extubation but his trachea was re-intubated after 2 minutes of apnoea, at a time when he must have been very lightly anaesthetised. He immediately became pulseless and florid pulmonary oedema developed with pink frothy sputum bubbling up the tracheal tube, although true cardiac arrest apparently did not occur. He was extubated after 9 hours of IPPV, having been given methylprednisolone and diuretics. He had no sickling on a blood film after the event and made a complete recovery.

Discussion

Many studies of anaesthetic mortality have been published in recent years.[1] Anaesthetic morbidity, which is more difficult to identify and define, has received less attention. Moreover, comparison between different series is limited by different populations, definitions of morbidity and geographical and time-related differences in anaesthetic practice.

Utting and colleagues,[2] for example, reported anaesthetic incidents which resulted in medicolegal consequences. These may not be directly related to the severity of morbidity in clinical terms and does not allow an assessment of overall morbidity frequency. A survey of nonfatal anaesthetic complications from Manitoba[3] reported an overall incidence of morbidity of 17.8% but included all unwanted consequences of anaesthesia, however minor they were.

A review similar to our own, of complications that resulted in admission to ICU, reported 110 admissions from 83 173 operations performed.[4] This rate of 1 in 756, approximately twice our own, includes significantly more cardiopulmonary arrests after operation than our series, for reasons which are not clear, but they may represent geographical differences in the provision of postoperative supervision of patients.

A 1986 prospective survey by Tiret and Hatton[5] of nearly 200 000 anaesthetics from 460 institutions in France provides an interesting comparison with our own work. Major complications occurred within 24 hours of an anaesthetic in 1 in 739 cases. A comparison of this rate with our own is limited by differences in definition. Our own choice of 'requiring ICU admission' as the criterion for entry to the study is, we think, not only a valid indicator of severity of morbidity but also has a useful application to the planning of resources for postoperative care. Our review should include most cases defined as intermediate and severe morbidity.[6]

All of our cases were admitted to the ICU within 12 hours of the end of the procedure. It is possible, however, that the anaesthetic contribution to later postoperative complications is less easily identified and some cases may be missed in this way.

The French study[5] demonstrates a preponderance of complications during recovery similar to our own. Interestingly, they report a much higher mortality from problems after anaesthesia compared with those which developed during maintenance. This was not the case in our own series and probably represents the difference in provision of care for the recovery period between our hospitals and theirs, where many patients were sent straight to the wards from theatre. The lowest mortality rates in both studies derive from the problems during induction; this may reflect either the nature of these complications or the intensity of vigilance and (or) monitoring during this time.

The seven-fold increase in the likelihood of complications after emergency procedures is worthy of careful note. Increased vigilance and help for the anaesthetist who performs during emergencies must be a *sine qua non* of provision by authorities.

The incidence of some conditions may have been affected by our selection criteria. The surprising absence of anaphylaxis as a cause for admission to ICU, for example, despite an incidence of about 1 in 5000 reported by Tiret *et al*;[5] may be explained by the obvious manifestation of these reactions and their favourable response to prompt therapy. Similarly, the number of cases of suxamethonium apnoea (incidence of homozygote 1 in 3000) may possibly be under-represented in our study since only a proportion of cases will require ventilatory support for longer than the period for which anaesthesia is required.

The difficulties in comparing and reconciling different studies is well illustrated by the variation in the nature of complications due to differences in clinical practice, as reported recently from Copenhagen.[7] The range of complications in that series follows the more widespread use of regional and neurolept anaesthesia, although they also report a surprisingly high incidence of transient suxamethonium bradycardia and asystole.

There is a noteworthy absence of cases with avoidable elements managed by senior house officers. This demonstrates that our most junior staff are protected successfully from the more difficult problems. The cases are otherwise spread in proportion to the numbers of staff in each grade.

The large group of patients admitted to ICU as a result of ventilatory inadequacy were elderly and had emergency laparotomy: many also had concurrent medical problems. The 11 patients in this category who had undergone emergency laparotomy represents 1.6% (11 of 700) of such cases. However, the data suggest that, if morbidity is to be further improved, emergency laparotomy in patients aged over 70 years should be followed by elective IPPV postoperatively rather than by an immediate trial of reversal of muscle relaxants and extubation.

The permanent neurological morbidity in two cases cost

£0.6 million when the cases eventually came to court. A figure equal to 13% of this was spent in the intervening period, both in the private and public sectors, on monitors which are either exclusively pulse oximeters or contain pulse oximeters. The sound investment sense of this is obvious.

We hope to report a reduced incidence of these problems when we review admissions for the 5 years after this study. This may be as a result of an increased awareness of the need for elective ventilation in some elderly patients after operation; superior monitoring facilities, including neuromuscular monitoring and the use of newer anaesthetic drugs.

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Mortality during intensive care after orthotopic liver transplantation

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Summary

The postoperative course of 335 adult patients who underwent orthotopic liver transplantation from 1968–1987 was reviewed retrospectively to identify patients who died in the intensive care unit and the causes of death. Forty-four percent of all deaths occurred in the intensive care unit. The mortality rate in the intensive care unit peaked in 1984 (48%), but decreased to 11% in 1987. The main causes for death in the intensive care unit were infection (55%) and haemorrhage (19%). The patients who died spent more time in the intensive care unit, had a longer period of tracheal intubation and received a larger intra-operative blood transfusion than patients who died in other locations.

Key words

Surgery; Transplantation.
Complications; death.

Orthotopic liver transplantation (OLT) was pioneered originally in the United States by Moore[1] and Starzl.[2] The first orthotopic liver transplant in man was performed by Starzl in 1963. The operation was first performed in the United Kingdom by Calne in 1968. It is now accepted as a therapeutic modality in end-stage liver disease, acute hepatic failure, inborn errors of metabolism and some liver tumours.[3]

Liver transplantation has been performed for 20 years in the joint programme between Cambridge and King's College Hospital. This retrospective analysis of the experience in the Cambridge/King's College series attempts to identify the major causes of mortality of patients during their stay in the intensive care unit and to provide information on the changing patterns of problems which have contributed to mortality.

Patients and methods

Three hundred and thirty-five adult patients (over 14 years of age) have had at least one liver transplant in the Cambridge/King's College Hospital series during the period from 1968 to the end of October 1987. These patients were grouped according to a chronological scheme: 41 patients from 1968–1975; 60 patients from 1976–1980; 45 patients from 1981–1983; and 27–60 patients from each of the years 1984 to 1987. These groups were chosen as being of comparable size. The numbers in the groups from 1984 onwards represent a major increase in transplant activity in this hospital. Paediatric liver transplantation (now an increasing part of our practice) is not included in this study

Indications for transplantation include chronic advanced hepatobiliary disease (CAHD) (biliary cirrhosis, chronic active hepatitis, alcoholic cirrhosis, cryptogenic cirrhosis and sclerosing cholangitis), acute liver failure (viral infection or poisoning), hepatic malignancy, inborn errors of metabolism (Wilson's Disease, alpha-1-antitrypsin deficiency, oxalosis) and the Budd Chiari syndrome. Surgical, anaesthetic and medical management is described in detail elsewhere.[4]

The place of death, time between operation and death, number of re-operations (if any), intra-operative blood transfusion, duration of stay in the intensive care unit (ICU) and the time of tracheal intubation were recorded for all patients who died. In the early part of the series intensive care measures such as artificial ventilation, tracheal intubation, catecholamine infusion and invasive monitoring were practised outside the ICU but, for statistical purposes, these patients are considered as if they had

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Table 1. Number (percentage of group) of deaths that occurred in the intensive care unit after orthotopic liver transplantation in seven time periods.

	1968–75	1976–80	1981–83	1984	1985	1986	1987
Number of patients	41	60	45	27	45	57	60
Age, years (SD)	41.2 (12.8)	40.4 (12.2)	39.7 (12.5)	37.4 (13.5)	40.5 (10.3)	41.4 (10.8)	39.8 (12.2)
Deaths in ICU (first admission)	9 (21%)	8 (13%)	12 (27%)	7 (26%)	6 (13%)	7 (12%)	5 (8%)
Deaths in ICU (readmission)	4 (10%)	5 (8%)	8 (18%)	6 (22%)	4 (9%)	3 (5%)	2 (3%)
Deaths in ICU (Total)	13 (31%)	13 (21%)	20 (45%)	13 (48%)	10 (22%)	10 (17%)	7 (11%)

received this therapy and died in the ICU. The main cause of death was obtained from the postmortem results (when available) or clinical data according to criteria used previously.[5] When possible, only one (the major) cause of death was identified for each patient. Multisystem organ failure (MOF) was recorded as a cause of death when failure of two or more major organ systems was present. There are predominantly three causes of multisystem organ failure in this group of patients; infection, or infarction, or rejection of the donor liver. Infection was coded as the cause of death if there was microbiological confirmation of an organism (culture or serology). Infarction of the liver is characterised by a marked increase in the serum concentrations of transaminases and prothrombin time and by sudden deterioration in the patient's condition. Rejection can be diagnosed histologically or by the response to pulsed bolus doses of steroids. Infection, infarction or rejection was coded as the primary cause of death if proven; multisystem organ failure was coded if no cause was identified. Renal failure was defined as the presence of a plasma creatinine concentration of > 217 μmol/litre or if there was a need for haemodialysis or haemofiltration. All other concurrent pathology present at the time of death was coded as an associated diagnosis. Student's t-test for unpaired data was used for statistical analysis where applicable.

Results

The numbers of patients who underwent OLT in each time period are shown in Table 1. One hundred and ninety-five of the 335 patients who have received OLT have died, 86 of them (44% of deaths) in the ICU. Table 1 also shows the numbers of patients who died in the ICU during either their first admission or on readmission. The age and gender of adult patients who underwent OLT did not change significantly during the study period although the number of orthotopic liver transplants performed each year has increased steadily, particularly since 1984. The mortality rates for both the first and subsequent ICU admissions have fallen significantly since 1984. The main indications for transplantation are shown in Table 2. Initially a large number of patients were transplanted for malignancy but this has now become an uncommon indication. Alcoholic cirrhosis had the lowest ICU mortality and Wilson's disease the highest although the small numbers prevent meaningful statistical analysis. The groups associated with the largest decreases in ICU mortality over the years are those with cryptogenic cirrhosis and malignancy.

The main causes of death are shown in Table 3. Infection has remained the most important cause of death (55%) with haemorrhage the next most common (19%). Associated causes of death are shown in Table 4. Multisystem failure and renal failure make up 60% of all contributory causes of death. Multisystem (which includes renal) failure became increasingly recognised in the period 1981–1983 as the commonest cause of death. During that period renal failure occurred in 45% of patients who died in the ICU, much more frequently than previously. The relationship of renal failure to ICU mortality and one-year survival can be seen from Figure 1. Many factors influence survival, but the development of renal failure in critically ill patients is associated with high mortality. The decreasing incidence of

Table 2. Primary diagnosis and mortality in the intensive care unit after orthotopic liver transplantation.

	1968–75			1976–80			1981–83			1984			1985			1986			1987		
	n	d	%	*n*	d	%	*n*	d	%	*n*	d	%	*n*	d	%	*n*	d	%	*n*	d	%
Biliary cirrhosis	3	1	(33)	14	3	(21)	16	9	(56)	7	5	(71)	7	3	(43)	19	4	(21)	16	2	(12)
Chronic active hepatitis	2	0	(0)	7	1	(14)	7	3	(43)	5	3	(60)	9	2	(22)	8	1	(12)	13	1	(8)
Alcoholic cirrhosis	1	0	(0)	3	0	(0)	1	0	(0)	1	0	(0)	1	0	(0)	1	0	(0)	5	1	(20)
Cryptogenic cirrhosis	5	2	(40)	5	2	(40)	3	1	(33)	2	1	(50)	3	1	(33)	6	1	(16)	5	0	(0)
Sclerosing cholangitis	—	—	—	2	0	(0)	2	2	(100)	—	—	—	4	1	(25)	4	0	(0)	4	1	(25)
Acute liver failure	—	—	—	—	—	—	—	—	—	3	1	(33)	1	0	(0)	5	2	(40)	6	0	(0)
Malignancy	29	10	(34)	21	4	(19)	11	3	(27)	6	1	(16)	13	1	(8)	5	0	(0)	5	1	(20)
Wilson's disease	—	—	—	—	—	—	1	0	(0)	1	1	(100)	2	1	(50)	1	0	(0)	1	1	(100)
Budd Chiari	—	—	—	5	2	(40)	3	1	(33)	2	1	(50)	3	0	(0)	3	1	(33)	1	0	(0)
Other	1	0	(0)	3	1	(33)	1	0	(0)	—	—	—	2	1	(50)	5	1	(20)	1	0	(0)

n, number of patients; d, ICU deaths.

Table 3. Main causes of death. Percentages refer to all deaths in that location in each time period.

	Deaths in ICU							Deaths outside ICU
	1968–75	1976–80	1981–83	1984	1985	1986	1987	1968–87*
Rejection	—	—	—	—	—	—	—	9 (9%)
Infarction	2 (15%)	1 (8%)	1 (5%)	1 (8%)	1 (10%)	—	—	6 (6%)
Infection	8 (62%)	7 (54%)	11 (55%)	8 (62%)	4 (40%)	4 (40%)	5 (71%)	30 (29%)
Haemorrhage	—	3 (23%)	3 (15%)	2 (15%)	3 (30%)	4 (40%)	1 (14%)	1 (1%)
Multisystem failure	1 (8%)	1 (8%)	4 (20%)	2 (15%)	—	1 (10%)	—	—
Recurrence	1 (8%)	—	—	—	—	—	—	30 (29%)
Biliary anastomosis	—	—	—	—	—	—	—	3 (3%)
Pulmonary embolus	—	—	—	—	—	—	—	3 (3%)
Gastrointestinal haemorrhage	—	—	1 (5%)	—	—	—	—	2 (2%)
Cerebral haemorrhage	1 (8%)	1 (8%)	—	—	1 (10%)	1 (10%)	1 (14%)	—
Other	—	—	—	—	1 (10%)	—	—	10 (11%)
Unknown	—	—	—	—	—	—	—	7 (7%)

*Excluding theatre deaths.

Table 4. Associated causes of death in patients who died in ICU. Percentages are related to ICU deaths for that period.

	1968–75	1976–80	1981–83	1984	1985	1986	1987
Rejection	1 (8%)	—	—	—	—	—	—
Infarction	—	1 (8%)	—	—	1 (10%)	1 (10%)	—
Haemorrhage	—	—	1 (5%)	—	—	—	1 (14%)
Multisystem failure	1 (15%)	2 (15%)	14 (70%)	6 (46%)	3 (30%)	2 (20%)	2 (28%)
Biliary problems	1 (8%)	2 (15%)	1 (5%)	—	—	—	—
Pulmonary embolus	2 (15%)	1 (8%)	—	—	—	—	—
Gastrointestinal haemorrhage	—	—	—	—	1 (10%)	—	—
Cerebral haemorrhage	—	1 (8%)	—	—	—	—	—
Renal failure	1 (8%)	—	9 (45%)	3 (23%)	1 (10%)	1 (10%)	1 (14%)
Cardiac failure	—	—	—	—	1 (10%)	1 (10%)	—
Disseminated intravascular coagulation	—	—	—	1 (8%)	—	—	—
Mesenteric thrombosis	—	—	—	—	1 (10%)	—	—
Miscellaneous	1 (8%)	—	—	1 (8%)	—	1 (10%)	—

renal failure in this group appears to be associated with a decrease in ICU mortality (except for 1987) and an increase in one-year survival.

Table 5 presents further information on patients who died during admission to the ICU or after discharge. There is no statistically significant difference in age or gender. The average number of days spent in the ICU was shortest for patients who survived but there was no statistically significant difference in duration of ICU stay between patients who died during their first ICU admission and those who died in their second ICU admission. Patients who died had a longer period of tracheal intubation and required more intra-operative blood transfusion. The incidences of the main causes of death for all patients are shown in Figure 2.

Discussion

The current one-year survival after OLT at this centre is 66%. This is higher than that reported by the European

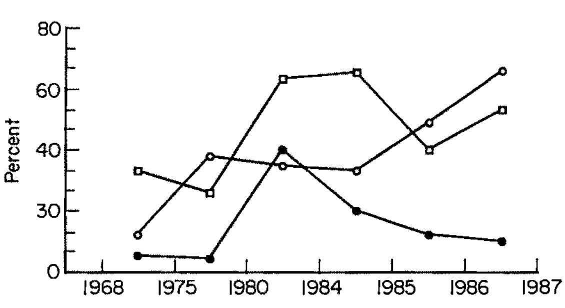

Fig. 1. The incidence of renal failure during the period of intensive care in patients who had undergone liver transplantation between 1968 and 1987. Mortality (as a percentage of all deaths) and one-year survival for the same period are shown also. —O—, one-year survival; —●—, renal failure; —□—, ICU deaths.

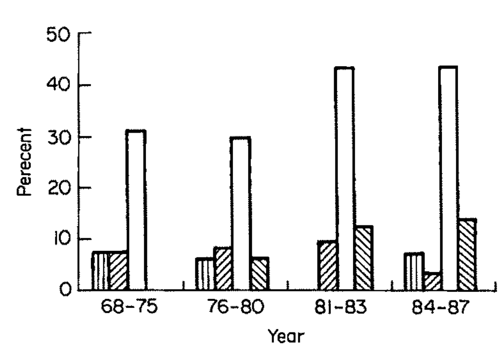

Fig. 2. Main causes of death after liver transplantation for the period 1968 to 1987. ▥, rejection; ▨, infarction; □, infection; ▧, haemorrhage.

Table 5. Risk factors associated with death after liver transplantation. Values are expressed as mean (SD).

	Deaths outside ICU ($n = 81$)*	Deaths during first admission to ICU ($n = 49$)	Deaths during readmission to ICU ($n = 30$)
Age	41 (12)	41 (10)	39 (12)
Sex	34 M, 47 F	25 M, 24 F	13 M, 17 F
Survival time (days)	350.7 (600.6)	11.6 (10.6)†	89.1 (136.2)†‡
ICU time (days)	6.1 (10.1)	11.6 (10.6)	10.9 (14.4)
Patient days	424	593	293
Intubation time (hours)	38.5 (78.9)	162.5 (200.4)†	124.2 (245.3)†
Intra-operative blood transfusion (litres)	8.1 (6.9)	19.6 (15.0)	9.8 (7.6)§
Total number of re-operations	43	34	16
Number of patients (% of total) re-operated	35 (43%)	24 (49%)	12 (40%)
ratio	1.2 : 1	1.5 : 1	1.3 : 1

*Excluding deaths in theatre.
†$p < 0.01$ (with respect to column 1).
‡$p < 0.01$, §$p < 0.05$. (with respect to column 2).

Transplant Registry[6] and compares well with other series.[7–9] Survival has improved steadily from 12% during the first 8 years up to 49% in 1985 and 66% in 1986. The percentage of deaths that occurred in ICU increased from 31% in the initial period to 48% in 1984 but since that time it has fallen each year and was 11% in 1987.

Major changes in the management of these patients occurred during the study period. Postoperative artificial ventilation for the first 24 hours was introduced in 1976; prior to this period patients were extubated at the end of operation and allowed to breathe spontaneously.[10]

The period with the maximum incidence of multisystem organ failure including renal failure (1981–1984) coincides with the introduction of cyclosporin A, given immediately after OLT, into clinical practice. This high incidence of renal failure caused a change in our therapeutic regimen. Cyclosporin is now administered 48 hours after the end of operation when the patient is haemodynamically stable and has adequate renal function. Further research into renal protection led us to introduce low-dose dopamine infusion as part of routine management. This resulted in a reduced incidence of renal impairment and less need for haemodialysis.[11] These changes and other more general measures (see below) were associated with reductions in the incidence of renal failure in patients who died in the period from 1981–1983 (45%), to 23% in 1984 and 10% in 1986. Simultaneously the one-year survival rates increased to 49% and 66% in 1985 and 1986 respectively (Fig. 1).

The major cause of death in the ICU continues to be infection, an experience reported also by others.[5] Its total incidence appears not to have changed with time; it remains the primary cause of death in 35–45% of all patients and contributes to 55% of all ICU deaths. The high incidence of infection is a reflection of the severity of pre-existing illness, the magnitude of the operation and the need for immunosuppression. Almost all of the deaths caused by haemorrhage, multisystem failure and cerebral problems occurred in the ICU. Half of the deaths associated with ischaemic damage to the donor organ occurred during the period of intensive care. No deaths attributed primarily to rejection, pulmonary embolism or biliary problems occurred in the ICU.

The volume of blood transfused intra-operatively appeared to be related closely to the ICU deaths that occurred early in the postoperative period (Table 5). The mean blood usage in patients who died in the ICU was 19.6 litres, more than reported by others;[12] this may reflect differences between the patient groups or techniques in the two centres. Periods of hypotension, residual peritoneal blood clot and tamponade of renal vessels by large amounts of blood[13] may lead to an increased risk of renal failure and infection. Patients who died in a subsequent ICU admission also had a larger blood loss than survivors.

The durations of stay in the ICU and of tracheal intubation were, as might be expected, longer in the nonsurvivors (on average more than 10 days and 120 hours respectively).

Less quantifiable aspects of postoperative intensive care concern the adoption of a management protocol in 1984, subsequently revised annually. Medical and nurse staffing levels have also improved during this time. We have not attempted to analyse the effects of these developments but others have shown that outcome in an ICU with adequate numbers of trained staff is better than in a unit with low staff numbers.[14] Other factors, not related directly to the intensive care these patients received, contributed also to the improved survival during the review period. Improvements in donor maintenance,[15] organ preservation[16] and antibiotic prophylaxis have occurred and there has been a change in the diagnostic groups transplanted. Originally a large number of patients were transplanted for malignancy. Unfortunately many of these patients developed recurrence of their tumour and it is not now a common indication for transplantation. Patients with malignancy are usually transplanted more rapidly than others with the result that they tend to be less unwell than those with end-stage liver disease and so pose fewer peri-operative problems, a feature demonstrated in their low overall mortality since 1975.

Eleven percent of patients died during the period of intensive care in 1987 despite improvements in management. Two major causes of mortality, infection and haemorrhage, require attention. It is of note that these have remained essentially unchanged during this period. Some of the deaths diagnosed as being related to infection may have

been caused by rejection and the fine balance between excessive immunosuppression which may result in infection and inadequate immunosuppression with a consequent increased risk of rejection requires careful management. Postoperative haemorrhage continues to be a problem and may represent a complication of surgery, derangement in the coagulation mechanism due to the pre-existing poor synthetic liver function or failure of synthetic function of the new liver.

Acknowledgments

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Postoperative analgesia for haemorrhoidectomy

A comparison between caudal and local infiltration

S. J. PRYN, M. M. CROSSE, M. S. C. MURISON AND F. P. McGINN

Summary

This study compared the analgesic effectiveness of local infiltration of bupivacaine with caudal extradural bupivacaine in the first 48 hours after haemorrhoidectomy. Surgical and anaesthetic protocol was rigidly standardised. The caudal group had significantly less pain in the first 6 hours after haemorrhoidectomy, and on first bowel opening, when compared to those who received local infiltration of bupivacaine. There was no significant difference between the two groups with respect to further analgesic requirements, complications, time to first bowel action, and duration of hospital stay. The definite advantage of caudal extradural bupivacaine for haemorrhoidectomy must be balanced against the rare but potentially serious complications associated with its use.

Key words

Pain; postoperative.
Anaesthetic technique; caudal.

Haemorrhoidectomy has been described as agonisingly painful[1] and has unfavourable notoriety in this respect. However, there is evidence that caudal epidural analgesia offers patients some benefits.[2-5] The study by Berstock[3] showed that bupivacaine caudal analgesia dramatically reduces the postoperative opiate requirement (by 79%) and halves the time to first bowel action (from 4 to 2 days). Presumably the two are related, since opiate drugs tend to cause constipation.

Local infiltration of bupivacaine can also provide good operative anaesthesia for haemorrhoidectomy,[6,7] and although to our knowledge there are no studies which demonstrate continued analgesia into the postoperative period, one can reasonably expect this to occur, since it does at other injection sites.[8]

Both methods of administration of bupivacaine are regularly used in our hospital to provide postoperative pain relief after haemorrhoidectomy. It is proposed that if the technique of local infiltration can be shown to be as effective as the already proven advantages of caudal analgesia, then this may be a safer, quicker and more reliable method. In addition, if one method provides superior analgesia compared to the other, then one might expect a greater opiate sparing effect, and hence a shorter time to bowel action and discharge from hospital. This would add the advantage of cheaper patient care.

Methods

Forty-four consecutive patients about to undergo elective haemorrhoidectomy under general anaesthesia by one of the two participating surgeons were randomly allocated into two groups of 22. Group 1 received caudal analgesia and Group 2 local infiltration analgesia with 0.25 ml/kg bupivacaine 0.5% as part of the anaesthetic technique. The only exclusion criteria were for those patients with specific contraindications to caudal extradural analgesia (e.g. coagulopathy, localised infection, pre-existing neurological deficit, patient refusal, and those with known hypersensitivity to local anaesthetic agents). All patients gave their informed consent before operation and approval for the study was obtained from the district ethics committee.

The surgical protocol was rigidly standardised. All patients had third-degree piles, or second-degree after failed Barron's band ligation, and they received an enema the day before surgery. The surgeon infiltrated around the haemorrhoids 0.25 ml/kg of a solution of either 1 in 200 000 adrenaline (Group 1), or bupivacaine 0.5% with 1 in 200 000 adrenaline (Group 2), after induction of general anaesthesia. Haemorrhoidectomy was performed by Milligan–Morgan ligation excision[9] and the wound dressed with a Jelonet pack which was left in place until the next morning. The patients were treated with regular aperients

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Table 1. Demographic details of patients; mean with (95% confidence limits).

	Group 1 (n = 22) Caudal	Group 2 (n = 22) Local infiltration
Age, years	54.8 (48.0–61.6)	58.2 (52.5–64.0)
Weight, kg	74.4 (67.6–81.1)	71.8 (67.8–75.8)
Males : females	12 : 10	11 : 11

after operation, and discharged from hospital when their bowels had opened and they were comfortable. Follow-up was at 6 weeks in the outpatient clinic.

The anaesthetic protocol was also standardised. Premedication consisted of temazepam 20 mg and metoclopramide 10 mg orally one hour before operation. Anaesthesia was induced with thiopentone 4–6 mg/kg and maintained with enflurane in nitrous oxide and oxygen. Those patients allocated to Group 1 then received a caudal extradural injection of 0.25 ml/kg bupivacaine 0.5% performed by one of the authors (M.M.C.) who has experience of many thousands of caudal injections. Analgesia was supplemented after operation with oral coproxamol (two tablets) on patient request, or intramuscular papaveretum if the pain was severe or the response to oral medication was unsatisfactory. This was at the discretion of the nursing staff.

The patients were assessed by one of us (S.J.P.) in a double-blind design after operation. Neither the patient nor the investigator knew which method of analgesia had been provided intra-operatively. Pain was assessed using a 100-mm visual analogue scale and a simple four-point scale (no, mild, moderate, or severe pain) shortly after awakening from the general anaesthetic and then at 3, 6, 12, 24 and 48 hours after operation. Assessments of pain during pack removal and during first bowel action were recorded by the patients immediately after the events. Analgesic consumption, the time to first bowel opening and discharge from hospital were also recorded, as was the incidence of side effects that could be attributed to the local anaesthetic administration.

The treatment groups were compared using statistical techniques of significance for rejection of the null hypothesis. Demographic data were analysed using the unpaired t-test (age and weight) or Chi-squared with Yates' correction

Table 2. 100-mm linear analogue pain scores (0 = no pain, 100 = worst pain imaginable) at various times after operation; shown as median; quartiles (range).

Time after operation	Group 1 Caudal	Group 2 Local infiltration
On awakening	1;* 0–39 (0–93)	25.5; 7–50 (0–90)
3 hours	3.5;* 0–32 (0–86)	24.5; 18–36 (2–88)
6 hours	5.5;** 0–24 (0–46)	28.5; 14–60 (4–71)
12 hours	26; 9–44 (0–63)	36; 13–45 (2–81)
24 hours	18.5; 10–44 (0–85)	18; 7–26 (1–47)
48 hours	19; 7–26 (0–72)	20.5; 7–38 (1–78)
Pack removal	37; 35–57 (0–99)	48.5; 37–72 (2–98)
First bowel action	32;* 15–66 (0–100)	52; 47–91 (2–100)

*$p < 0.05$; **$p < 0.01$.

(sex). Visual analogue pain scores were not distributed normally so were analysed using the Mann–Whitney U test and are presented as a median with quartiles and range; the four-point pain scores were analysed using Fisher's exact probability test. Fisher's exact test was also used for comparison of analgesic usage and complication rate between the two groups. The times to first analgesic usage, bowel opening and discharge were compared using the unpaired t-test. Calculated mean values are quoted with their 95% confidence limits wherever appropriate.

Results

The demographic data for the two randomly allocated treatment groups are shown in Table 1; there was no significant difference between them, which confirms that they were reasonable samples from the same population.

The linear analogue pain scores presented in Table 2 demonstrate the huge range of scores within each treatment group. Nevertheless, when the median values are compared it is apparent that the caudal group had significantly less pain than the infiltration group for the first 6 hours after operation. The caudal group also fared better during first bowel opening. The four-point pain scores presented in Table 3 confirm that the caudal group had more patients who recorded no pain in the first 6 hours after operation. Supplementary postoperative analgesic requirements were not significantly different between the two treatment groups (Table 4).

Table 3. Pain scores (four-point scale) at various times after operation, shown as the number of patients in each group who recorded either no, mild, moderate or severe pain.

Time after operation	Group 1 Caudal (n = 22)				Group 2 Local infiltration (n = 22)			
	No	Mild	Moderate	Severe	No	Mild	Moderate	Severe
On awakening	14*	2	3	3	6	8	6	2
3 hours	13**	4	3	2	2	13	6	1
6 hours	11**	7	4	0	2	10	9	1
12 hours	4	10	8	0	2	10	8	2
24 hours	2	14	5	1	3	14	5	0
48 hours	5	10	7	0	1	13	7	1
Pack removal	2	6	9	5	0	4	12	6
First bowel action	2	7	7	6	0	3	11	8

*$p < 0.05$; **$p < 0.01$.

Table 4. Analgesic requirements in the first 48 hours after operation (95% confidence limits).

	Group Caudal (n = 22)		Group 2 Local infiltration (n = 22)	
Required papaveretum	3		6	
Required coproxamol	19		22	
Mean time to first analgesia, hours	7.3	(4.5–10.1)	7.2	(5.1–9.3)
Mean number of requests for analgesia (per patient per 48 hours)	3.6	(2.7–4.5)	3.6	(2.9–4.4)

Table 5. Time to first bowel action and length of hospital stay. Mean with (95% confidence limits).

	Group 1 Caudal		Group 2 Local infiltration	
Time to first bowel action/hours	36.7	(28.9–44.5)	42.5	(32.7–52.3)
Time to discharge home/hours	61.7	(54.1–69.4)	64.1	(56.1–72.1)

Similarly there was no significant difference between the two groups when time to first bowel action, time to discharge home (Table 5), and postoperative complication rate (Table 6) were compared. All but one of the four patients who complained of transient leg weakness, shown in Table 6, were able to walk normally at 4 hours; the other (in the caudal group), complained of a weak ankle and could not walk for 11 hours after operation. There were no intra-operative complications attributable to the method of analgesia.

Discussion

This study clearly demonstrated that haemorrhoidectomy patients who received intra-operative caudal bupivacaine experienced significantly less pain for up to 6 hours after operation than a similar group of patients who received a local infiltration of bupivacaine. Over 50% of the caudal group experienced no pain at all during this time as compared to 9% of the local infiltration group. The duration of analgesia after caudal bupivacaine 0.5% of 7.3 hours, as estimated by the time to first request for supplementary analgesics, is similar to that found in studies after haemorrhoidectomy: 6.3 hours,[2] 9.6 hours,[4] and 8 hours.[5] However there was no significant difference between the proportions of patients in each treatment group who experienced 'satisfactory' postoperative analgesia (no pain or mild pain only).

The reliability of provision of postoperative analgesia was disappointing, since 27% of the caudal group and 45% of the local infiltration group experienced moderate or severe pain in the first 6 hours after operation. In most hands one can expect a failure rate of 5–10% after caudal injections.[10]

Pybus[4] showed that the addition of morphine to a caudal local anaesthetic block (lignocaine) improved the efficacy of analgesia after haemorrhoidectomy; however Boskovski[11] reported no advantages of adding morphine to bupivacaine caudals in similar patients. It would be interesting to see if the addition of small amounts of opiates to bupivacaine caudals could reduce the incidence of blocks, which appear to be working intra-operatively but leave patients suffering pain almost immediately after operation (23% in our study).

Table 6. Postoperative complication rate.

	Group 1 Caudal (n = 22)	Group 2 Local infiltration (n = 22)
Urinary retention	0	0
Urinary hesitancy	8	7
Leg weakness	3	1
Haemorrhage	1	1
Infection	0	0
Permanent neurological deficit	0	0

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The *in vitro* buffering capacity of soluble paracetamol

D. C. MILLS

Summary

The capacity to neutralise gastric acid was investigated in three over-the-counter paracetamol preparations. Panadol (Winthrop Laboratories) showed a buffering capacity equal to that of existing antacids because of the agents used to make it dissolve. This property, together with its mild analgesic action, widespread availability and palatability, may make it a useful agent for acid aspiration prophylaxis.

Key words

Complications; aspiration.
Analgesics; paracetamol.

Antacids are an important component in the provision of effective prophylaxis of acid aspiration syndrome and an intragastric pH above 2.5 is quoted as acceptable.[1] A previous study[2] of the *in vitro* buffering capacity of sodium citrate, magnesium trisilicate mixture (MMT), and Alka Seltzer Effervescent, an 'over-the-counter' preparation available in the United States that contains sodium bicarbonate, potassium bicarbonate and sodium citrate, found all three preparations effective in opposing the change in pH on addition of hydrochloric acid. Further investigations of Alka Seltzer Effervescent were suggested because of the problems of the short shelf life of sodium citrate and the particulate nature and difficulty with mixing of MMT. However Alka Seltzer Effervescent is not widely available in this country.

Observations of soluble paracetamol tablets together with compositional data from the manufacturers suggested that paracetamol was encouraged to dissolve by an effervescent compound similar to Alka Seltzer Effervescent. This study was performed to evaluate and compare the buffering capacity of various commercial soluble paracetamol compounds, using a similar method to that of Murrell and Rosen.[2]

Methods

The following proprietary paracetamol compounds were investigated: Panadol, paracetamol soluble (Winthrop Laboratories); Hedex Soluble (Sterling Health); Disprol Paediatric (Reckitt and Coleman).

The pH measurements were made with a Beckmann model 3500 digital pH meter with the slope set between 95 and 100%. Calibrations before and after each set of titrations were made with BDH Colourkey buffer solutions 19240 to pH 7.00 and solution 19239 to pH 4.00 and were found to be within 0.02 units for each series.

Titration was carried out with hydrochloric acid 1 mol/litre added rapidly in 1- or 2-ml aliquots from a burette into the solution which was stirred by a magnetic stirrer. This acid solution is 10 times stronger than gastric acid, but it provided convenient volumes with which to work. The pH reading was allowed up to 2 minutes to settle to a stable level and the reading was then taken. This was necessary with the first 10 ml of added acid, with further acid the readings stabilised within seconds.

Two tablets of Panadol that each contained 500 mg paracetamol were dissolved in 50 ml water. This volume was chosen after preliminary investigations showed that this was the minimum volume necessary to allow rapid dissolution. One sachet of Hedex Soluble that contained 1 g paracetamol was dissolved in 50 ml water. Two sachets of Disprol Paediatric that contained 240 mg paracetamol in 10 ml yellow syrup was diluted to 20 ml of solution to allow the pH meter to be completely covered. The initial results showed Panadol to be the best buffer, so it was further investigated.

The effect of exposure to air. The buffering capacity of the mixture was measured at intervals of 5, 10, 15 and 30 minutes after the tablets were dropped into the water.

The effect of mixing. Panadol solution 50 ml was slowly and carefully trickled down a pipette onto the surface of 100 ml hydrochloric acid 0.1 mol/litre. The solution was

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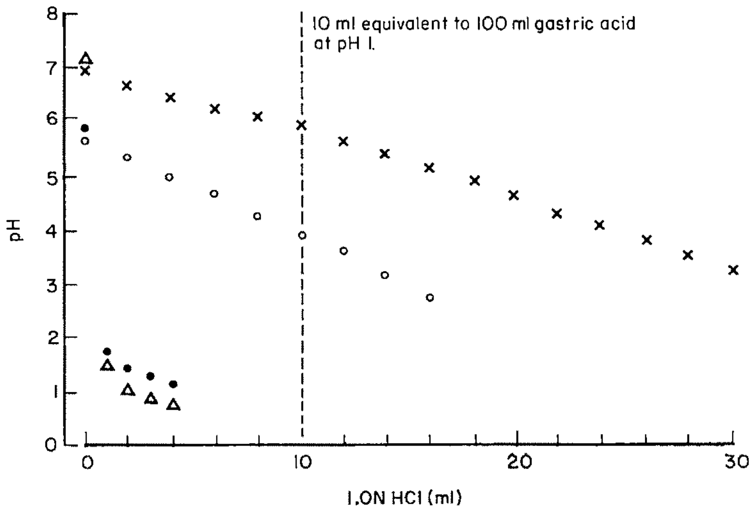

Fig. 1. Comparison of the pH changes on addition of acid to the study solutions. ×, 50 ml Panadol solution; ○, 50 ml Hedex solution; △, 20 ml Disprol solution; ●, 50 ml water.

observed and pH measurement of the top and bottom parts of the solution were performed after 5 minutes.

The appearance of the solution. The appearance of the solution was noted after the addition of 50, 70, 90 and 100 ml hydrochloric acid 0.1 mol/litre to 50 ml Panadol solution.

Results

Panadol acted as a buffer to prevent the change in pH of the solution on addition of acid. Hedex Soluble was less effective, while Disprol Paediatric was less effective than water (Fig. 1). Ten millilitres of hydrochloric acid 1 mol/litre in this experiment is equivalent to 100 ml normal gastric acid, shown on the figure, and provides a guide to the efficacy of the solution in neutralising the acid. The intercept for Panadol is pH 5.8. More than 300 ml of gastric acid would have to be present to reduce the pH to less than 3 and therefore it is likely that it would raise the intragastric pH above 2.5 under normal conditions. The intercept for Hedex is 3.9 and therefore there is much less safety margin.

Effect of exposure to air. Figure 2 shows that standing open to air has little effect on the buffering capacity of Panadol.

The effect of mixing. It was difficult to make two separate layers when the Panadol solution was added to hydrochloric acid 0.1 mol/litre. Moderate effervescence occurred immediately and this presumably aided diffusion and even mixing of the solution. The pH at all levels of the mixture was 5.72.

The appearance of the solutions. Panadol takes 2.5 minutes to stop effervescing when dissolved in 50 ml of water. It forms a particulate solution. The particles form a layer on the base of the container in the absence of continuous mixing. However, on addition of 50 ml hydrochloric

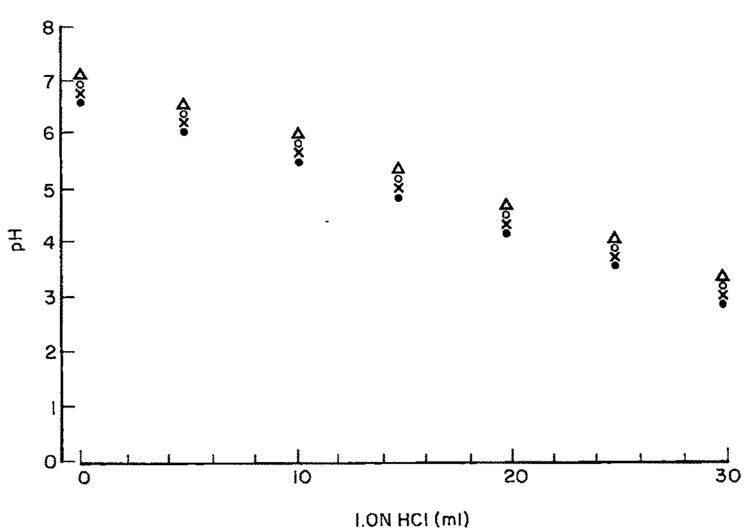

Fig. 2. Buffering capacity of 50 ml Panadol solution on standing open to air for 5 (●), 10 (○), 15 (×) and 30 (△) minutes.

acid 0.1 mol/litre, further moderate effervescence occurs, the solution becomes much clearer and leaves a very small amount of precipitate. Further acid 0.1 mol/litre up to 150 ml total volume causes the precipitate to dissolve completely.

Discussion

This study shows that proprietary soluble paracetamol products have potentially useful buffer properties. The addition of two tablets of Panadol dissolved in 50 ml water to 100 ml gastric acid would produce a solution of pH 5.8. The equivalent pH value for 20 ml MMT is approximately 7.0, 30 ml sodium citrate 0.3 mol/litre is 4.4, and two Alka Seltzer Effervescent tablets in 20 ml water is 5.3.[2] Hedex is a less powerful buffer, while Disprol, a commonly used paediatric preparation, has no buffering capacity at all.

Patients with a gastric pH of less than 2.5 and a gastric volume of more than 0.4 ml/kg are generally thought to be at risk of acid aspiration syndrome.[1] Antacid solutions increase pH but also increase gastric volume.[3] Particulate solutions such as MMT may cause pulmonary lesions as a result of aspiration of antacid particles.[4] Extra risk factors include outpatients[5] and children,[6] as well as pregnancy, obesity and emergency surgery.

Panadol dissolved in 50 ml forms a particulate solution. However the particles disappear on the addition of further volume or acid, to form a clear solution which mixes easily. Clear fluids have a very rapid gastric emptying rate,[7,8] and 50 ml water has been used with oral premedication without an increase in gastric volume.[9] It is unlikely therefore that problems due to the volume or the particulate nature of the solution should occur in the clinical setting.

Paracetamol has been used in studies of postoperative analgesia. Dental extraction procedures have frequently been used and paracetamol has been found to be better than placebo.[10,11] It has also been found to be useful after laparoscopy.[12]

One tablet of Panadol contains 500 mg paracetamol, 1500 mg sodium bicarbonate (17.9 mmol), 925 mg citric acid (4.8 mmol), 30 mg sodium carbonate (0.3 mmol), together with preservatives and binders. There is a total of 18.5 mmol sodium per tablet and therefore a two-tablet dose contains approximately twice the amount of sodium than 20 ml sodium citrate (18 mmol), and 30 ml MMT (18.9 mmol).[2] This may be of importance in patients with cardiac or renal impairment.

Panadol is in everyday use in hospitals and the community therefore palatability should not be a problem. Carbon dioxide production in the stomach with a theoretical rise in intragastric pressure should be kept to a minimum, as long as it is given after effervescence ceases. It has a long shelf life and is easily stored, unlike sodium citrate or MMT. Its widespread availability may be an advantage in wards which do not routinely stock these other antacids.

Further studies are warranted, to investigate these properties of Panadol, in view of the double benefit of acid aspiration prophylaxis and analgesic effect, particularly in outpatients and those patients having minor surgery.

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Transtracheal illumination for optimal tracheal tube placement

A clinical study

S. MEHTA

Summary

The tracheas of 420 adult patients were intubated using the tip of a lighted stylet placed inside the lumen of the tracheal tube, just proximal to the tube cuff. The maximum point of transillumination was visible just distal to the cricoid cartilage, with proper cuff positioning. The lighted stylet was also introduced into the oesophagus to see whether transoesophageal illumination could be demonstrated. The intensity of transillumination was measured using a grading system. The distance between the bevel end of the tracheal tube and the carina was determined with a fibreoptic bronchoscope. Tracheal transillumination was graded as excellent in 81% of patients and as good in 19%, when the overhead lights were dimmed and cricoid pressure was applied. Transoesophageal illumination could not be demonstrated in any patient. The mean distance between the tip of the tracheal tube and the carina varied between 3.7 and 4 cm. Transtracheal illumination is a simple, effective and reliable method that can be used during intubation for the recognition of optimal tube placement.

Key words

Intubation tracheal; technique.

A review of various anaesthetic-related morbidity and mortality statistics indicates that unrecognised oesophageal intubation remains a problem.[1] An analysis of anaesthetic accidents, leading to liability claims in the United Kingdom from 1977 to 1982, listed oesophageal intubation as a main cause of accidents leading to death and neurological damage.[2] Undiagnosed bronchial intubation is associated with hypoxaemia and increased airway resistance, and undue pressure of the tracheal tube cuff in the subglottic region can lead to unilateral vocal cord paralysis.[3]

The two most reliable techniques to determine proper tube placement when cords are not visible involve capnography and fibreoptic bronchoscopy. Both these techniques require the use of expensive equipment, which is not universally available in anaesthetic rooms in most hospitals in the UK. There is thus a need for a simple, inexpensive, yet reliable technique, which can be used in any location in the hospital where intubation is performed. The present study was designed to examine whether transtracheal illumination can be used to confirm correct tracheal tube placement.

Methods

Four hundred and twenty adult patients who were to undergo elective general surgical and gynaecological procedures gave consent to participate in the study. A set of three procedures was carried out in each patient, after induction of anaesthesia and muscle relaxation. All patients were intubated with a 7.0–8.0-mm internal diameter Franklin Medical tracheal tube with a Sensive large volume, low pressure cuff.

The tip of the lighted stylet was placed inside the lumen of the tube, just proximal to the tracheal tube cuff, before intubation was attempted (Fig. 1). Maximum point of transillumination was visible just distal to the cricoid cartilage, with proper cuff positioning. The effect of cricoid pressure and dimming of overhead lights on transtracheal illumination was noted (Fig. 2), and the intensity of transillumination measured using a grading system: nil, poor, good and excellent. The intensity of the room light was recorded with a Thorn EMI lightmeter LM4. The tracheal tube was secured and the cuff inflated to a no-leak volume.

The lighted stylet was then introduced into the oesophagus to see whether transoesophageal illumination could be demonstrated. Finally, with the head in the neutral position the fibreoptic bronchoscope was introduced into the tracheal tube and the distance between the bevel end of the tracheal tube and the carina measured.

Results

The details of the patients studied are given in Table 1; their mean weight was 60.2 (range 40–107) kg. Transtracheal illumination data are shown in Table 2. In 340 patients (81%) the transillumination was graded as excel-

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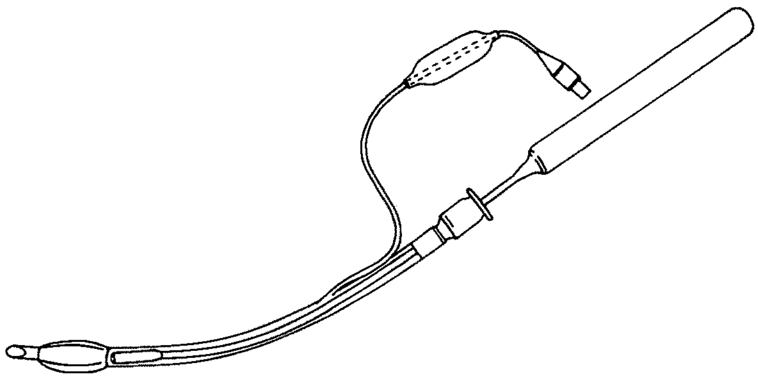

Fig. 1. The tip of the lighted stylet placed just proximal to the cuff.

lent and in 80 patients (19%) as good when the overhead lights were dimmed and cricoid pressure applied. Application of cricoid pressure alone or dimming of overhead lights increased the incidence of successful transtracheal illumination. However, transoesophageal illumination could not be demonstrated in any patient when the lighted stylet was in the oesophagus with the overhead lights turned down and the application of cricoid pressure.

Table 3 shows the mean distance between the tip of the tracheal tube and the carina in both male and female patients. The mean distance varied between 3.7 and 4 cm when the tracheal tube was positioned with the help of the lighted stylet.

Discussion

An illuminated introducer for a tracheal tube was first described by Sir Robert Macintosh in 1952,[4] although orotracheal intubation using transillumination of neck tissues to guide tube placement was first mentioned in the literature in 1959.[5] Foster and Ducrow described the use of transillumination with a fibreoptic light or lighted stylet as an aid to blind intubation.[6,7] The technique then remained relatively obscure for a number of years. Recently, guided orotracheal intubation, using a lighted stylet, was compared with both direct vision orotracheal intubation and the blind nasal approach.[8,9] The stylet is also used to position the proximal end of the cuff of a plastic tracheal tube just below the cricoid cartilage.[10]

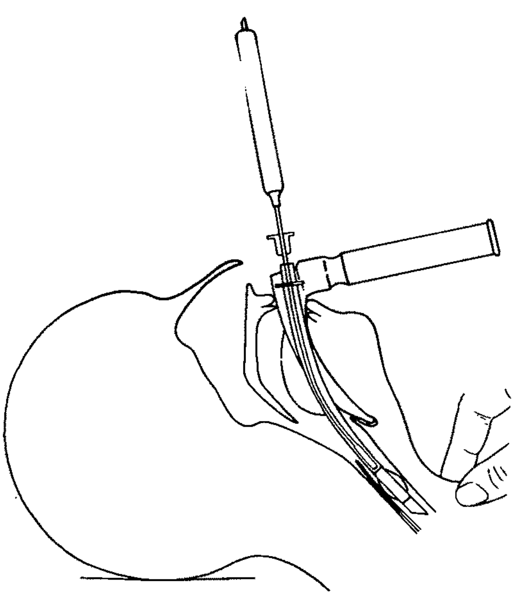

Fig. 2. Cricoid pressure applied during the correct placement of the tube at laryngoscopy.

A lighted stylet was mainly used in the past to facilitate intubation in difficult cases, but not as a method or test to confirm the correct placement of a tracheal tube. The technique of transtracheal illumination not only allows confirmation of the position of the tube in the trachea, but also enables one to place the cuff just below the cricoid cartilage and ensure that the tip of the tracheal tube lies in the middle third of the trachea. Transtracheal illumination was observed in all patients, with the overhead lights turned down and the application of cricoid pressure. Under similar circumstances, however, transoesophageal illumination could not be demonstrated. This study included average adults and several obese individuals with short, thick necks. A number of patients were of Asian and West Indian origin.

Transillumination or transmission of light through tissues depends on several factors, such as thickness, compactness, density and light absorption characteristics of the tissue, wavelength, quality and intensity of light, proximity of the tissue to the source of light and the ambient lighting condition in the room. The study shows the transillumination is more obvious in a darkened room and can be easily demonstrated. However, indirect lighting can be permitted and this would maintain sufficient illumination to observe the patient. The intensity of ambient lighting in this study, measured at the anterior part of the neck, varied between 20 and 70 Lux when the overhead lights were dimmed. The application of cricoid pressure by approximating the tracheal wall to the source of light and

Table 1. Demographic details (SD).

Total	420
Male : female	175 : 245
Mean age, years	41.6 (12.6)
Range	17–84
Mean weight, kg	60.2 (13.2)
Range	40–107

Table 2. Transtracheal illumination data.

Grades of transtracheal illumination	Overhead room lights on	Overhead lights on and cricoid pressure	Overhead lights dimmed	Overhead lights dimmed and cricoid pressure
	Illuminance or lighting intensity 350–600 lux		Illuminance 20–70 lux	
Excellent, *n*	0	2 (0.47%)	0	340 (81.0%)
Good, *n*	1 (0.2%)	83 (19.8%)	78 (18.6%)	80 (19.0%)
Poor, *n*	20 (4.8%)	110 (26.2%)	80 (19.0%)	0
Nil, *n*	399 (95.0%)	225 (53.6%)	262 (62.4%)	0

stretching the soft tissues in front of the neck, promoted transillumination.

Neck flexion may result in bronchial intubation if the tracheal tube is placed far beyond the cords. It is recommended that the tip of the tracheal tube be placed in the middle third of the trachea with the neck in the neutral position.[11] This study confirmed that transtracheal illumination can be used for correct tube placement, so that the tip of the tracheal tube remains 3.7 to 4 cm above the carina.

Transtracheal illumination is a simple, safe, effective and reliable method that can be used routinely during intubation for optimal tube placement. This method is particularly useful to confirm the position of the tube in the trachea in cases of difficult intubation, when the larynx is either difficult to visualise or when there is difficulty in manoeuvring the tube through the larynx. The position of the tube in the trachea can be frequently rechecked in the ICU environment, where tube movement is a recurrent problem, and will reduce the need for repeated chest X rays. The complication of unilateral vocal cord paralysis can be avoided by placing the cuff below the subglottic region of the larynx. The disadvantage of the transillumination technique is the need to dim the room lights and its possible failure in the presence of midline neck tumours or swellings.

It has been suggested that any test to confirm tracheal tube placement should fulfil certain criteria, such as, it should work in difficult situations. It needs to be unequivocal and must never give a false sign.[12] To these, other criteria can be added; the test should be simple, rapid and reliable. It should require neither the use of elaborate equipment nor any previous experience. The technique of tracheal illumination fulfils all these criteria and it is hoped that this technique will reduce markedly the frequency of unrecognised tube malposition, accidental bronchial intubation and unilateral vocal cord paralysis.

Table 3. Distance between the tip of tracheal tube and the carina. Values expressed as mean (SD).

Sex	*n*	Distance in cm
Female	245	3.7 (0.4)
Male	175	4.0 (0.5)

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Anaesthesia, 1989, Volume 44, pages 973–974

CASE REPORT

Effect of limb tourniquet on cerebral perfusion pressure in a head-injured patient

P. R. ELDRIDGE AND S. WILLIAMS

Summary

Thirty percent of patients with severe head injury also have significant extracranial injuries. Treatment for these injuries should not be allowed to jeopardise the brain which is more susceptible to damage in these circumstances. A case is presented in which significant decrease in cerebral perfusion pressure occurred consequent upon use of a lower limb tourniquet.

Key words

Brain; intracranial pressure.
Equipment; tourniquets.

Brain function and cellular viability are dependent on adequate cerebral blood flow (CBF).[1] This in turn is dependent on cerebral perfusion pressure (CPP), the difference between mean arterial pressure (MAP) and intracranial pressure (ICP). Autoregulation in the normal individual maintains a constant blood flow over a range of blood pressures. However, in the presence of head injury, autoregulation may be defective and the brain more susceptible to ischaemic damage. Cerebral perfusion may be at risk both from raised ICP from the head injury and from decreased MAP due to haemorrhage from other injuries.[2] In addition ICP increases when arterial $P\text{CO}_2$ is allowed to rise, and a variety of drugs, including inhalational anaesthetic agents may also increase ICP. It is important that the effects of treatment for extracranial injuries do not prejudice the brain. We present a case in which routine management of a limb injury resulted in poor cerebral perfusion, and suggest ways to avoid this problem.

Case history

A 17-year-old male pedestrian was struck by a car. He was instantly rendered unconscious and on admission shortly afterwards was flexing nonpurposefully to pain, with no eye-opening or verbal response, although breathing spontaneously with normal blood gases. His pupils were small and reacting. CT scan showed loss of basal cisterns, with effacement of the sylvian fissure and cortical sulci, which suggested raised ICP. Medical management of raised ICP, which consisted of tracheal intubation, sedation, hyperventilation and hypothermia, was instituted. ICP was monitored in order to assess the effects of this treatment. Extracranial injuries consisted of a comminuted fracture of the surgical neck of the left humerus and tears to the medial collateral and anterior cruciate ligaments in the left knee. Orthopaedic opinion was that the humerus should be fixed internally and the knee ligaments explored and repaired under tourniquet to improve the surgical field and reduce blood loss.

The patient was taken to the operating theatre where a subarachnoid (Richmond type) screw was inserted. End-tidal $P\text{CO}_2$, heart rate, ICP and radial arterial pressure were recorded continuously. CPP was computed and recorded simultaneously. The humerus was fixed internally, and the knee ligaments then repaired in an exsanguinated limb under tourniquet.

Figure 1 shows the record from time of application to release of tourniquet (approximately 90 minutes). MAP was 70 mmHg when the tourniquet was applied (A) and increased over the next few minutes to 105 mmHg (B). Simultaneously the mean ICP rose from 8 to 18 mmHg. CPP increased from 62 to 87 mmHg as the increase in MAP exceeded that in ICP. Thiopentone 250 mg was given to reduce the ICP. Blood pressure, ICP and CPP returned to their previous values. Table height was changed for surgical comfort at (C), which altered the pressure obtained from both transducers by the same amount (5 mmHg); subsequent values were corrected for this artefact. CPP was unaffected, as the transducers moved together. The tourniquet was released at (D). End-tidal $P\text{CO}_2$ increased from 2.6

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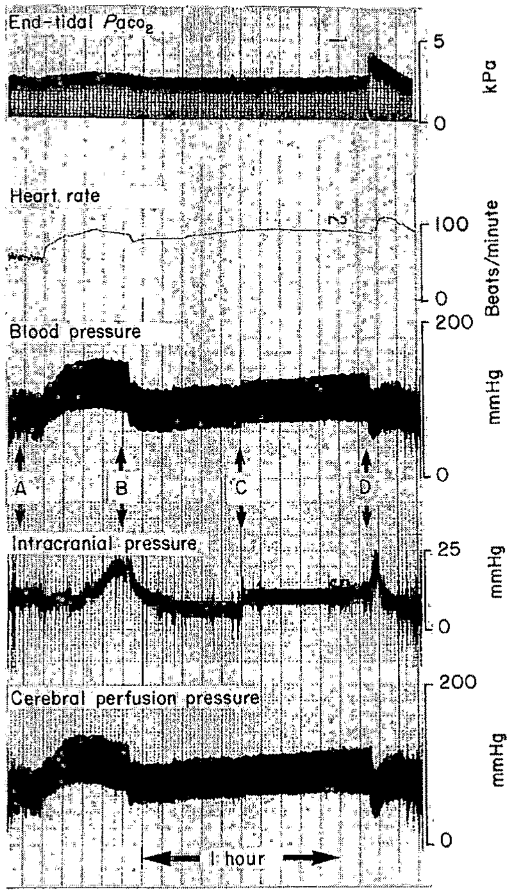

Fig. 1. Recordings of end-tidal P_{CO_2}, heart rate, blood pressure, ICP, CPP. Event (A) application of tourniquet; (B) administration of thiopentone 250 mg; (C) change in height of operating table; (D) tourniquet release.

to 4.6 kPa and this change was accompanied by an increase in ICP to 15 mmHg and a decrease in MAP to 65 mmHg. CPP decreased to 50 mmHg. Further thiopentone was given to reduce ICP, and 10 minutes later these variables returned to their previous values.

This regimen was withdrawn progressively without further increase in ICP, after 72 hours of hyperventilation with cooling. He had made full neurological recovery at follow-up 6 months later.

Discussion

There were no sequelae in this case but the margin for error was small. A CPP of 50 mmHg was recommended as the minimum safe value.[3,4] This value was reached in this instance. The consequences could have been life threatening if further problems had occurred such as heavy bleeding on release of tourniquet. Monitoring allowed early recognition and prompt treatment of the condition.

The increase in MAP and ICP on application of the tourniquet may be due to volume loading as the leg is emptied of blood. A more likely explanation is that anaesthesia was relatively light at this point; this was suspected at the time of operation. This is consistent with the effect on blood pressure that occurred 5 minutes after application of the tourniquet, and took 20 minutes to reach its peak. The increase in ICP was even more delayed.

The increase in ICP on release of the tourniquet is probably due to increased Pa_{CO_2}, and the decrease in blood pressure both to loss of volume into the limb and release of metabolites from the ischaemic limb.

These changes are predictable. Peroperative monitoring of end-tidal P_{CO_2} and MAP, preferably with record-showing trends, will identify the risk and show efficacy of treatment in patients with multiple injuries, including mild head injury, without the need for formal ICP monitoring. Such a facility should be available in all hospitals. Patients should be volume-replete before tourniquet release; moderate though not extreme hyperventilation (to approximately 3.2 kPa) will also be helpful.

Note added at proof. We have since observed a similar effect in another patient after release of an upper limb tourniquet and another case was reported this year (*Anaesthesiology* 1989; **71:** 294–5).

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Anaesthesia, 1989, Volume 44, pages 975–977

CASE REPORT

Anaesthesia for patients with mitochondrial myopathy

A. M. BURNS AND M. P. SHELLY

Summary

The anaesthetic management for an infant with mitochondrial encephalomyopathy due to fumarase deficiency is described.. Mitochondrial myopathies may produce skeletal and cardiac muscle abnormalities, central nervous system effects and metabolic problems. The solutions to the anaesthetic problems posed by these patients are discussed.

Key words

Muscle, skeletal; myopathy.
Enzyme; fumarase.

The term mitochondrial myopathy is used to describe a group of disorders of varying aetiology which have structural mitochondrial abnormalities on skeletal muscle biopsy. They may present at any age, and may affect skeletal muscle, cardiac muscle and other organs including the liver, brain and kidneys. They are termed mitochondrial encephalomyopathies when both the skeletal muscle and the brain are involved.

We present the anaesthetic management of an infant with mitochondrial encephalomyopathy due to fumarase deficiency. Fumarase is an enzyme in the tricarboxylic (K-rebs) cycle. Fumarase deficiency as a cause of mitochondrial myopathy is rare and only three cases have been previously described.[1,2] The implications of anaesthesia in patients with mitochondrial myopathy are discussed.

Case history

A 6-week-old female infant who weighed 3.5 kg, presented for muscle, skin and open liver biopsy under general anaesthesia. She was the second child of Indian parents, who were cousins; her elder brother was 2 years old and healthy. She was delivered vaginally after an uncomplicated pregnancy after labour was induced for postmaturity. Her birth weight was 3.64 kg and Apgar scores were 5, 8 and 9 at 1, 5 and 10 minutes respectively. She was admitted after delivery to the special care baby unit after a cyanotic episode secondary to a patent ductus arteriosus, which subsequently closed spontaneously. Further examination and investigation showed jaundice with hepatomegaly, hypoglycaemia, hypotonia, and a cerebral ultrasound scan demonstrated an absent corpus callosum with mild communicating hydrocephalus. Investigation into the cause of these abnormalities revealed fumaric acidaemia and fumaric aciduria presumed to be secondary to fumarase deficiency. She was discharged after 5 weeks in the special care baby unit with persistent jaundice, poor feeding and weight loss, and hypotonia. She was readmitted for the biopsy procedures to confirm the presumed diagnosis, to determine the extent of the biochemical abnormality, to assess prognosis and to offer the parents genetic counselling. She was jaundiced with hepatomegaly and mildly hypotonic. There was no clinical evidence of cardiac failure, although there was a persistent systolic murmur audible at the left sternal edge. Biochemical investigations included sodium 134 mmol/litre potassium 4.1 mmol/litre, urea 2.2 mmol/litre and creatinine 26 μmol/litre. Liver function tests demonstrated a bilirubin of 122 μmol/litre, alkaline phosphatase 1375 IU/litre and aspartate transaminase of 64 IU/litre. Serum lactate was normal and there was no evidence of a metabolic acidosis before operation.[3] Haemoglobin was normal at 137 g/litre and a clotting screen demonstrated a normal prothrombin ratio of 1.1 and a prolonged activated partial thromboplastin time ratio of 1.38. A sickle cell screening test was negative. Forty millilitres of fresh frozen plasma were given immediately before operation to correct the clotting abnormality; one unit of blood was available.

No premedication was given. A 5% dextrose infusion was started 4 hours beforehand to avoid hypoglycaemia

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after fasting. Monitoring with a pulse oximeter, an ECG, a precordial stethoscope and of rectal and peripheral temperatures was established on arrival in theatre. The blood glucose before induction was 4–6 mmol/litre.

Venous access was established and anaesthesia induced intravenously with thiopentone. Further monitoring intraoperatively included an oesophageal stethoscope in place of the precordial stethoscope, automatic noninvasive blood pressure monitoring, inspired oxygen concentration analyser and a peripheral nerve stimulator. Oxygen saturation remained between 98 and 100% throughout the anaesthetic; both temperatures remained between 36 and 37°C, and with a train-of-four nerve stimulation pattern at 2Hz, four twitches of clinically equal amplitude were present at all times. An arterial blood sample taken after induction of anaesthesia, to investigate the possibility of a metabolic acidosis, in association with the hepatic dysfunction and the myopathy, revealed a pH 7.21, $P\text{CO}_2$ 5.4 kPa, $P\text{O}_2$ 29 kPa and base excess −4.2 mmol/litre on $F\text{IO}_2$ 0.5.

General anaesthesia was maintained initially with isoflurane and nitrous oxide in oxygen ($F\text{IO}_2 = 0.5$) with spontaneous ventilation through an Ayre's T-piece. The concentration of isoflurane was increased to 2.5% in 100% oxygen before the open liver biopsy, and tracheal intubation was performed with a plain tracheal tube size 3.0 mm whilst the child breathed spontaneously. Ventilation was then controlled manually through the Ayre's T-piece with isoflurane and nitrous oxide in oxygen. Two millilitres 0.25% plain bupivacaine was infiltrated into the wound prior to closure. The infant was extubated in the left lateral position when awake, and monitored overnight on an apnoea blanket. The dextrose infusion was continued until feeding was re-established. Postoperative follow-up revealed no complications of the procedure and no deterioration in the biochemical indices of hepatic and renal function. She was discharged 2 days after operation. Histology of the biopsy specimens and detailed biochemical tests subsequently confirmed the diagnosis of mitochondrial encephalomyopathy secondary to fumarase deficiency.[3]

Discussion

The mitochondrial myopathies have a variable clinical picture; the symptoms and signs may be nonspecific, and the patients may not present until late in life.[4] The different clinical patterns include involvement of the extraocular muscles alone, a group with generalised limb weakness often associated with marked fatiguability, and a multisystem disorder with cardiac and central nervous system involvement in addition to the myopathy.[5,6] There is no constant pattern of inheritance.

Abnormalities of the shape and number of mitochondria in skeletal muscle biopsy are a nonspecific feature and may occur in various muscle diseases. A diagnosis of mitochondrial myopathy is made if such changes are the main feature of an abnormal skeletal muscle fibre. More recently, specific biochemical defects in mitochondrial function have been recognised, but no link has been established between such defects and the associated clinical features.[5,6] Several specific problems face the anaesthetist when presented with a patient with a known or suspected diagnosis of mitochondrial myopathy.

The possibility of a myopathy must invite two important questions; is there an increased susceptibility to malignant hyperthermia (MH) and, if a muscle relaxant is to be used, which relaxant would be the most appropriate and in what dosage? A predisposition to MH on exposure to a triggering agent is associated with various myopathies; the most well established association is with central core disease.[7] There has been one report of a child with mitochondrial myopathy who developed signs of MH after induction of general anaesthesia.[8] This was treated promptly but there were no details of subsequent *in vitro* tests of muscle contractility specific for MH. In the absence of more specific information it seems wise to consider the possibility of MH and monitor both core and peripheral temperatures throughout general anaesthesia.

Care should be taken with the administration of muscle relaxants if their use is judged to be clinically appropriate. Suxamethonium may trigger MH.[8] There is no specific information about the use of nondepolarising agents in patients with mitochondrial myopathy, so the minimum required dose should be used and the effects and duration of action of the neuromuscular blocking agent monitored with a peripheral nerve stimulator. The trachea was intubated with the patient breathing spontaneously in our case and relaxation was adequate to allow open liver biopsy. Had greater relaxation been necessary, we planned to use atracurium rather than increase the concentration of isoflurane which might have resulted in significant cardiovascular depression. This seemed to be the most appropriate agent in view of the patient's hepatic dysfunction.

There may be hepatic and renal dysfunction pre-operatively and this requires careful evaluation to determine its extent. Clotting abnormalities should be corrected with fresh frozen plasma before surgery and blood cross-matched. Anaesthetic agents which may adversely affect hepatic or renal function or which have a significant renal excretion should be avoided where possible. The pharmacokinetics of drugs such as opioids may be altered and their duration of action may be prolonged. The use of drugs such as isoflurane, which has minimal hepatic metabolism and renal excretion, and atracurium, which undergoes Hoffman degradation, would seem most appropriate.

Metabolic problems may include hypoglycaemia secondary to hepatic dysfunction and pre-operative fasting. Blood sugar should be monitored and a glucose infusion started to avoid intra-operative hypoglycaemia. Lactic acidosis may be associated with myopathy particularly related to fasting and exercise, and may exacerbate a metabolic acidosis due to hepatic and renal dysfunction. The use of a glucose infusion may help to avoid acidosis secondary to fasting, but arterial blood pH estimation may detect the development of a metabolic acidosis during long procedures. Hypoxia and hypotension should be avoided and tissue perfusion well maintained. Signs of a developing acidosis include hypotension and tachycardia, and an increased respiratory rate in a patient who is breathing spontaneously. The mainstay of management of a metabolic acidosis requires identification and treatment of the underlying cause.[9] The role of bicarbonate in the management of lactic acidosis remains controversial.[9]

Cardiac muscle may be affected resulting in a cardiomyopathy. Cardiovascular assessment is important and any cardiac failure should be adequately treated before anaesthesia. The anaesthetic technique should aim to avoid myocardial depression and cardiovascular instability, and appropriate monitoring should be employed.

Finally, the extent of any cerebral dysfunction and any focal neurological deficit should be carefully recorded. Patients with a significant deficit may have an increased sensitivity to all sedatives and to opioid analgesics, and these should be carefully titrated to achieve the desired response. The use of opioids may produce significant respiratory depression in an infant, so the use of bupivacaine infiltrated locally may be an alternative means to provide satisfactory analgesia.

Mitochondrial myopathies are a group of disorders with a variable clinical picture and an underlying mitochondrial metabolic defect. They may present several challenges to the anaesthetist, especially when there is multisystem involvement. This case report describes an anaesthetic technique which may be of value to other anaesthetists if faced with this rare condition.

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Anaesthesia, 1989, Volume 44, pages 978–981

APPARATUS

Ventilator disconnexion alarm failures

The role of ventilator and breathing system accessories

S. J. PRYN AND M. M. CROSSE

Summary

Pressure-sensitive ventilator disconnexion alarms do not always alarm during disconnexion of a discharging compliance ventilator such as Manley Blease, unless accurately adjusted. High flows during disconnexion result in significant pressure generation caused by outflow resistance of catheter mounts, heat and moisture exchangers, capnometer cuvettes, and angled connectors; this may lead to alarm failure because of incorrectly adjusted pressure alarm limits. The exact position of the disconnexion is critical and if the alarm's pressure sensor is placed in either the inspiratory or expiratory limb of the ventilator it makes no difference to its correct function. Nine different heat and moisture exchangers were compared in the same breathing system. Those with 15-mm male connectors generate the highest pressures on disconnexion (1.1 kPa). It is suggested that the low pressure alarm limit is set only marginally below the peak inspiratory pressure, and that it is readjusted for every patient and after every change in ventilation. Most importantly, the alarm should be shown to be functional by a trial disconnexion at the tracheal tube.

Key words

Equipment; disconnexions.

An alarm that does not work is more dangerous than no alarm at all. However, ventilator disconnexion alarms must be correctly adjusted in order to function usefully. Pressure-sensitive ventilator disconnexion alarms work by monitoring pressure in the breathing system through a T-piece and comparing this to a preset low pressure alarm limit. The device assumes that a disconnexion has occurred and alarms, if the pressure does not increase above the low alarm limit within a certain time, (often 10 seconds).

Previous reports of ventilator alarm failures have described pressures generated proximal to the disconnexion, owing to high gas flows across ventilator tubing[1] and catheter mount resistances.[2] Their recommendations were either to place the alarm in the expiratory limb of the ventilator breathing system or avoid using pressure-sensitive alarms.

We are fortunate in our department in that we have recently acquired pressure-sensitive ventilator disconnexion alarms (Blease 73103) for all our anaesthetic machines. However, during a random check of 17 different theatres, in which adult patients were mechanically ventilated, a trial disconnexion between catheter mount and tracheal tube resulted in only two of the alarms correctly identifying the hazard; whilst if the heat and moisture exchanger (HME) was removed from the system, or if the disconnexion was made at the patient-end of the ventilator tubing, then all the alarms were correctly set off. It seemed that both the exact point of disconnexion and the presence of an HME was critical to alarm function. The increasing use of breathing system accessories such as HMEs and capnometer cuvettes in modern anaesthesia thus presents an additional disconnexion hazard if this effect is not understood.

We have measured the pressures that are generated proximal to a disconnexion using a variety of ventilators, HMEs and a capnometer cuvette in this laboratory study. This will enable a more realistic and safe approach to adjusting ventilator disconnexion alarms.

Methods

Five different ventilators were adjusted to supply a tidal volume of 650 ml over a 1.5-second inspiratory time, using standard 22-mm female and 15-mm female connectors with a corrugated catheter mount, and a 15-mm right-angle connector (Fig. 1 for breathing system diagram). Peak pressures were measured in the inspiratory limb from the ventilators during simulated disconnexion at the tracheal tube using a pressure transducer and pen recorder (Hewlett Packard 79342A/78172A/1290/A). Disconnexions were repeated with a Portex Thermovent 1200 heat and moisture exchanger, and a Hewlett Packard capnometer cuvette in the system. The ventilators tested were: Manley Blease, Manley Servovent MS, Manley Pulmovent MPT, Penlon Nuffield 200, and Penlon Oxford. The Manley Blease was tested at bellows weights of 1.0, 1.5, 2.0, 2.5, 3.0, and 3.5 kPa.

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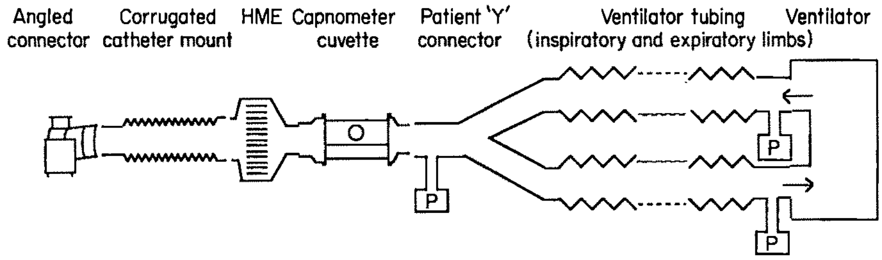

Fig. 1. Breathing system used to measure peak pressures during simulated disconnexions from various ventilators both with and without accessories such as HMEs and capnometer cuvettes. P represents the sites at which pressure was measured.

To demonstrate the site of outflow obstruction when the Manley Blease ventilator is used, disconnexions were made not only at the point of attachment of the tracheal tube but also between each separate component of the breathing system. In addition, the pressure transducer was placed at three different points in the breathing system that corresponded to the positions at which a disconnexion alarm can be placed: at both the inspiratory and expiratory ports of the ventilator, and at the patient 'Y' connector. The Manley Blease ventilator was adjusted to a tidal volume of 650 ml and a bellows weight for 2.0 kPa.

The effect of nine different HMEs, used both dry and moist, was also determined, using the same breathing system and monitoring equipment and a Manley Blease ventilator adjusted to a tidal volume of 650 ml and a bellows weight for 2.0 kPa. HMEs tested were: Pall Ultipor BB50T, Vitalograph Vapour Condenser, Siemens-Elma Servo 150, Engström Edith, Siemens Servo 153, Portex Thermovent 1200, Icor Vent-Aid IC101, Gibeck Humid-Vent 1, and MIH Mallinckrodt Inline. Peak pressures were recorded in the inspiratory limb from the ventilator during simulated disconnexions from the tracheal tube with the HMEs dry. The HMEs were then moistened by incorporating them in a breathing system for 30 minutes, through which a test lung was ventilated across a heated water humidifier held at 37°C. Simulated disconnexions were then repeated incorporating these moistened HMEs.

Results

Peak pressures likely to be detected by a monitor in the inspiratory limb during patient disconnexion from various ventilators are shown in Table 1. The Manley Blease generated the highest pressure at 0.7 kPa without, and 1.0 kPa with, a dry Thermovent 1200 HME. The other ventilators could only generate pressures of 0.25 kPa or less.

Figure 2 shows that most of the pressure generated by the Manley Blease ventilator during disconnexion arises from the resistance to flow in the catheter mount (0.4 kPa), with smaller amounts added by the HME (0.3 kPa), capnometer cuvette (0.15 kPa), right-angled connector (0.1 kPa), and ventilator tubing (0.05 kPa). There was no significant difference, to the nearest 0.1 kPa, in the peak pressure recorded when the pressure sensor was in different limbs of the Manley Blease system (inspiratory and expiratory limbs, and patient 'Y' connector).

Table 2 shows the pressure generated during patient disconnexion from the Manley Blease when the make of HME is varied. The highest pressures were recorded using HMEs that incorporated a 15-mm male connector: 1.05 kPa was generated using a dry Icor Vent-Aid. The pressures generated using moist HMEs were higher than when dry, but the effect was only slight, (an extra 0.05 kPa for the Icor).

The effects of varying the bellows weight on the Manley Blease are shown in Table 3: the peak pressure during disconnexion rises progressively from 0.5 to 1.45 kPa dependent upon the bellows weight. This is associated with a shortening of the inspiratory flow time, and hence a greater gas flow (up to a maximum of 108 litres/minute).

Discussion

The increasing use of HMEs and capnometer cuvettes, together with use of catheter mounts and angled connectors in this country, necessitates a more careful approach to the use of pressure-sensitive disconnexion alarms, if alarm failure and hence patient damage is to be avoided. This is

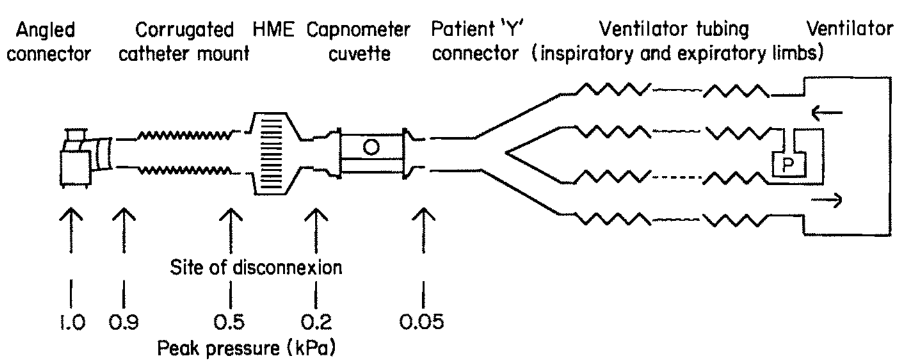

Fig. 2. Peak pressures (kPa) recorded in the ventilator inspiratory limb during disconnexion at various sites in the breathing system using the Manley Blease ventilator.

Table 1. Peak pressures (kPa) recorded in the ventilator inspiratory limb during disconnexion at the tracheal tube using different ventilators.

	Peak pressure (kPa)	
	Without HME	With HME
Manley Blease	0.7	1.0
Manley Servovent MS	0.15	0.2
Manley Pulmovent MPT	0.2	0.25
Penlon Nuffield 200	0.05	0.15
Penlon Oxford	0.15	0.25

Table 2. Peak pressures (kPa) recorded during disconnexion at the tracheal tube using a Manley Blease ventilator and different HMEs.

	Peak pressure (kPa)	
	Dry	Moist
Pall Ultipor BB50T	0.9	0.9
Vitalograph Vapour Condenser	0.8	0.8
Siemens-Elma Servo 150	0.9	0.9
Engström Edith	0.7	0.7
Siemens Servo 153	0.8	0.85
Portex Thermovent 1200	0.9	1.0
Icor Vent-Aid IC101	1.05	1.1
Gibeck Humid-Vent 1	0.9	1.05
MIH Mallinckrodt Inline	1.05	1.05

especially important if a discharging compliance ventilator is used (e.g. Manley Blease, East Radcliffe).

The Manley Blease ventilator, unlike the others tested, shortens its inspiratory flow time on disconnexion to 0.4 seconds (when set to a tidal volume of 650 ml and a bellows weight for 2.0 kPa) and so increases the inspiratory flow from 0.43 litres/second to 1.6 litres/second (i.e. 26 to 98 litres/minute). At this flow small resistances, not normally noticeable, will generate significant pressures because of outflow obstruction (up to 1.1 kPa with a moist Vent-Aid HME in circuit). This situation is exacerbated if higher bellows weights are used, as the peak flows and pressures generated are even greater (108 litres/minute and 1.45 kPa respectively).

All the HMEs tested added to the outflow obstruction on disconnexion. Those with 15-mm male connectors generated the highest pressures (Vent-Aid 1, Humid-Vent 1, Inline and Thermovent 1200). Slightly less pressure was generated by the Pall and Servo 150 (sponge); while least of all was generated by the Vitalograph (nondisposable), Engström Edith and Siemens Servo 153; the latter two

Table 4. Quoted resistance of moist HMEs as a pressure drop across them (kPa) at various flows.

	Litres/minute		
	30	60	90
Pall Ultipor BB50T	0.054	0.108	—
Vitalograph Vapour Condenser	0.018	—	—
Siemens-Elma Servo 150	0.05	0.1	—
Engström Edith	0.05	0.14	—
Siemens Servo 153	0.12	0.15	—
Portex Thermovent 1200	0.03	0.095	0.155
Icor Vent-Aid IC101	0.1	0.28	—
Gibeck Humid-Vent 1	0.11	0.3	—
MIH Mallinckrodt Inline	0.11	—	—

incorporate their own lightweight catheter mount. HME resistances quoted in the literature at 30 and 60 litres/minute seem modest (Table 4),[3,4] but these are misleading, because flows of 100 litres/minute and more can be expected during disconnexion when using a Manley Blease ventilator.

Shribman[1] has suggested placing the disconnexion monitor in the expiratory limb of the Manley ventilator, to bypass the pressure generated that resulted from outflow obstruction in the ventilator tubing. Our results suggest this would not be useful since most of the outflow obstruction occurs in the catheter mount and HME; furthermore the commonest site of actual disconnexion is at the tracheal tube.[5] A better site for the monitor would be at the tracheal tube, but the connecting adaptors are cumbersome and are not generally used at this site.

Ventilator disconnexion alarms can only provide a major increase in safety when adjusted correctly. The low pressure alarm limit is critical: this must be set above the pressure that can be generated by high flows through breathing systems and accessories, as demonstrated here. We endorse the recommendation of Myerson and his colleages[6] that the low pressure alarm limit be set only marginally below the maximum inspiratory pressure used for the ventilation of each individual patient; this is commonly around 1.2 kPa with anaesthetised patients. For absolute safety the alarm must be shown to be functional by a controlled trial disconnexion for each patient, at the connexion with the tracheal tube. This is a simplification of previous recommendations which suggested testing the alarm by controlled disconnexion at all critical joints before a breathing system is used.[7–9]

Lawrence[10] suggested, in a recent symposium on monitoring and patient safety, that the low pressure threshold should be restricted to an absolute minimum of about 0.8–

Table 3. Peak pressure (kPa) and estimated mean flow rate (tidal volume/inspiratory time) during disconnexion at the tracheal tube using different bellows weights with the Manley Blease ventilator (HME in the system).

Bellows weight (kPa)	Peak pressure (kPa)	Inspiratory flow time (seconds)	Estimated mean flow (litres/minute)
1.0	0.5	0.54	72
1.5	0.65	0.48	81
2.0	0.85	0.44	89
2.5	1.0	0.40	98
3.0	1.2	0.38	103
3.5	1.45	0.36	108

1.0 kPa. This seems sensible, although it would still not ensure a foolproof system, since we have demonstrated higher pressures generated during disconnexion.

Continued vigilance is necessary when using pressure-sensitive ventilator alarms particularly with ventilators similar to the Manley Blease. The alarm must not be relied upon unless it has been correctly adjusted for each individual patient, and has been demonstrated to be functional during a trial disconnexion at the tracheal tube. Furthermore any alteration in ventilator setting may necessitate readjustment of the alarm.

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Anaesthesia, 1989, Volume 44, pages 982–983

APPARATUS

Oropharyngeal volumes

G. N. NEWCOMBE AND W. J. RUSSELL

Summary

The volume of the mouth and pharynx was measured in 20 cadavers and ranged from 25 to 202 ml with a median value of 87 ml, a mean of 90.7 ml and a geometric (logarithmic) mean of 78.3 ml. The volume that encompasses 99% of the normal population is estimated as 215 ml if the assumption of a normal Gaussian distribution is made, or 349 ml if a log-normal distribution is assumed. Collection containers in suction equipment intended for removing vomit from the pharynx and mouth in adults should have a useable volume of at least 500 ml.

Key words

Anatomy; pharynx.
Equipment; suction machines.

Vomiting is of great concern to the anaesthetic and intensive care community, both because it is a common accompaniment of narcotic pain relief and general anaesthesia and because it is a major hazard in the patient with obtunded reflexes. There are British, Australian and International standards related to suction equipment, but there are only meagre data to support some aspects of the standards' requirements. This study was undertaken to find out the volume of the normal adult pharynx, which could then be a guide to the minimum size of the collection container in suction equipment intended for oral and pharyngeal suction.

There is no special need to minimise the size and weight of equipment in operating theatres with fixed installations. However, all aspects of size and weight deserve careful attention in field operations outside the hospital, where equipment may have to be carried considerable distances and passed through a relatively small opening, such as a car window.

The stomach may vary widely in size, but this is not the critical volume since most vomit will be expelled from the mouth. Suction equipment should relieve the risk of airway obstruction if it can clear the mouth and pharynx. A container with a volume several times greater than the pharyngeal volume would be helpful with multiple vomits, although one with a volume less than that of the pharynx would be useless since even a single vomit could not be managed. Thus, knowledge of the volume of the adult human mouth and pharynx is necessary to make informed decisions for suction equipment intended for pharyngeal suction.

A search of the literature failed to provide any information, so we measured the volume of the oral cavity and pharynx in 20 adults.

Methods

Recently deceased patients were examined immediately before autopsy. They were placed supine and their tracheas intubated with an 8-mm plastic tracheal tube with a soft cuff. The cuff was positioned just below the vocal cords and inflated with 15 ml air to achieve a firm seal. Dentures, if in place, were removed. Tap water in aliquots was then slowly instilled into the patient's mouth with a 20-ml syringe until the water began to drip from the mouth. The total volume was recorded, as was the subject's age, sex, height and weight. All volumes were corrected for the volume of the tube in the pharynx. This was estimated to be 7 ml when the 22-cm mark was at the mouth.

Results

Twenty cadavers were studied, 11 male and nine female. Five further subjects were unable to be intubated because

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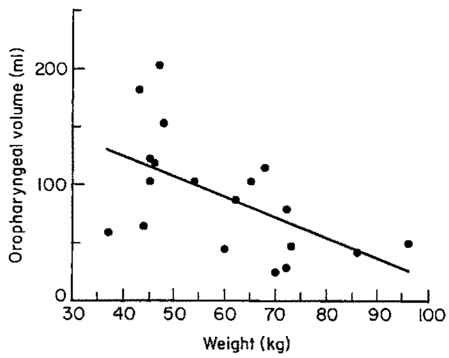

Fig. 1. Illustration of the relationship between body weight and oropharyngeal volume. The volume decreases linearly with body weight as the equation Vol = 196–1.77 weight $r = 0.57$.

of rigor mortis. The ages of the 20 subjects ranged from 17 to 86 years with a mean age of 68.2 years. Weight varied from 37 to 96 kg, and height from 145 to 178 cm.

Pharyngeal volumes ranged from 25 to 202 ml, with a median of 87 ml and a mean of 90.7 ml (SD 48.8) (Fig. 1). The geometric (logarithmic) mean volume was 78.3 ml. There was a moderate negative correlation between weight and volume ($r = -0.57$, $p < 0.02$), but there was no significant correlation between height and volume ($r = 0.09$). Similarly, there was no significant association between oropharyngeal volume and the sex of the subject. The mean volume for males was 99.5 ml (median 102 ml, SD, 61.0 ml) and for females 79.9 ml (median 79 ml, SD, 27.4 ml) T = 0.89.

Discussion

This study reveals the oropharyngeal volume to be surprisingly variable. It may be argued that this volume in cadavers may differ from that in life, although there are ethical difficulties in pouring water into the mouth and pharynx of live subjects. The rigidity of the cheek muscles could be expected to reduce the oropharyngeal capacity with rigor mortis. However, it would not be likely to change substantially the volume of most tissues such as the tongue and larynx. Thus our values may be a slight underestimate of the true volume, but should give a guide.

Oropharyngeal volume appears not to be closely related to height, and for weight there was a nonsignificant negative correlation with volume. This is surprising since we expected that a taller and heavier person would have a greater volume in his (her) mouth and pharynx. The male mean volume was higher than that of the females, although both had large standard deviations and the difference between the two means was not significant. Possibly further studies on greater numbers would confirm the trend towards a larger male pharynx, but it does not seem to be a major determinant of oropharyngeal volume.

It is difficult to ascertain the distribution of the underlying population of oropharyngeal volumes on these small numbers. The simplest assumption would be a normal Gaussian distribution. To cover 99% of the population on this basis it can be seen that the minimum effective suction collection container volume should be 215 ml. However, this gives negative values for two and three standard deviations below the mean. The mean value is 78.3 ml with a 2 SD spread of 24.5 to 249.6 ml or a 2.58 SD spread of 17.6 to 349 ml if a log-normal distribution is used, which corrects the skewed distribution and avoids negative values. Thus a 500-ml container would cover 99% of the population, assuming the log-normal distribution. A full mouth and pharynx could be cleared at least twice if a 500-ml collection container is used for 95% of the population, using either distribution.

At least one device currently marketed has a nominal volume of only 350 ml. The current standard for medical suction in the USA (ASTM F960-1986) makes no mention of a minimum collection container volume. Similarly, although collection containers used in hospital suction are required by the Australian Standard, AS2120-1977, to have a minimum volume of 1200 ml, and the British Standard BS4199 specifies a similar volume, there is no standard specification for suction equipment intended for emergency use outside the hospital. The international standard for medical suction which is currently being drafted by subcommittee 8 of the International Standards Organization Technical Committee 121, specifies a minimum useable volume of 500 ml for all situations. The standard also specifies a test for equipment intended to aspirate pharyngeal vomit, by suctioning of 200 ml of a simulated vomit mixture. It might be appropriate to increase the test volume to 250 ml in order to exceed the volume of 95% of the adult population.

Acknowledgments

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Anaesthesia, 1989, Volume 44, pages 984–985

Forum

Assessing the position of the tracheal tube
The reliability of different methods

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Summary

Various methods have been developed to confirm proper intubation of the trachea. This blind, randomised study evaluates some of these quantitatively and qualitatively. Forty patients had both their trachea and oesophagus intubated. A procedure that included auscultation of the upper abdomen and lungs was 100% reliable independent of which tube was ventilated. Auscultation of the lungs resulted in a wrong conclusion in 15% (6–30, 95% confidence limits) of the cases when the oesophagus was ventilated: the sounds were misinterpretated as normal breath sounds. Suction on the tubes with a 60-ml syringe was also a reliable test. Other methods assessed were observation for condensation of water vapor, and abnormal movements of the upper abdomen; these were unreliable.

Key words

Intubation, tracheal; technique.

The question whether the trachea has been intubated arises several times a day. Procedures to check this with certainty are widely reported.[1-13] Checking procedures have to be reliable, easily applied, and inexpensive to achieve widespread acceptance.

The reliability of several procedures was addressed in a blind, randomised clinical trial, which analysed some of the tests used when tracheal intubation and oesophageal intubation were performed.

Methods

The protocol for the study was approved by the Ethics Committee of Copenhagen City. All patients gave voluntary, informed consent. Forty patients (ASA grade 1 or 2) scheduled for gynaecological laparotomy were studied. Patients with oropharyngeal, tracheal, oesophageal or gastric diseases were excluded. All patients received as premedication, diazepam (0.2 mg/kg) orally. Administration of fentanyl 0.1 mg and pancuronium 0.01 mg/kg was followed by anaesthesia induced with thiopentone. Suxamethonium (1.5 mg/kg) preceded orotracheal intubation, performed with a Mallinckrodt Hi-Lo tracheal tube. The cuff was inflated until an airtight seal was obtained and the necessary pressure recorded. Anaesthesia was maintained with nitrous oxide and halothane in 33% oxygen. A similar tube was then inserted into the oesophagus and its cuff inflated to the same pressure as in the tracheal tube. The head and neck of the patient and the tubes were then covered with an opaque drape. The anaesthetic was changed to halothane in 100% oxygen. An investigator was shown one of the tubes after randomisation and asked to auscultate the epigastrium, the right and left axilla. A 'tidal' volume was delivered at each point, and the investigator indicated the position of the tube, and described the sounds heard at the axillae. A judgement of abdominal movement was also made at this stage. A change to the tracheal tube took place if the 'ventilated' tube was oesophageal. After another 3 minutes ventilation with 100% oxygen and halothane the investigator performed suction by syringe on a randomly presented tube, as described by Wee.[9] Free aspiration should lead to the conclusion that the tube is in the trachea, while resistance to aspiration should indicate that the tube is in the oesophagus.

A test was performed to observe whether condensation appeared at the tube mount or not, to indicate correct or oesophageal intubation. All the tests were performed twice in each patient using different observers.

Results

Twelve anaesthetists and 10 nurse anaesthetists participated as investigators. The position of the tube was correctly determined by auscultation of the epigastrium, the right and left axilla in all 80 tests, 40 times in the trachea and 40 times in the oesophagus.

The sounds in the axillae, when the oesophageal tube was ventilated, were recorded as normal breath sounds in six out of 40 tests (15% (6–30, 95% confidence limits)) and led to a false conclusion about the position of the tube. Ventilation of the tracheal tube gave normal breath sounds in the axillae in all cases. Movements of the epigastrium were judged normal in 36 (90% (76–97, 95% confidence limits)) out of 40 cases when the oesophageal tube was ventilated. The oesophageal detector device described by Wee correctly identified the position of the tubes. Tracheal

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Table 1. Investigators' conclusion on tube position.

Method of detection	Right, number (%)	Wrong, number (%)	Tube position
Auscultation of epigastrium,	40 (100)	0	Oesophagus
right and left axilla	40 (100)	0	Trachea
Auscultation of	34 (85)	6 (15)	Oesophagus
right and left axilla	40 (100)	0	Trachea
Suction with the oesophageal	40 (100)	0	Oesophagus
detector device	40 (100)	0	Trachea
Water vapour condensation	6 (15)	34 (85)	Oesophagus
on expiration	40 (100)	0	Trachea
Abdominal movement	4 (10)	36 (90)	Oesophagus
	40 (100)	0	Trachea

intubation allowed aspiration of more than 50 ml of gas free of resistance. Attempts to aspirate air from an oesophageal tube proved impossible.

Condensation was always observed in the transparent tube when the tube was in the trachea during expiration. However, when the tube was in the oesophagus condensation occurred 34 times (85% (70–94, 95% confidence limits)) in 40 cases. The results are summarised in Table 1.

Discussion

Unrecognised intubation of the oesophagus is a frequent cause of death or cerebral damage in anaesthesia.[14–16] This investigation evaluated some easy and inexpensive methods to determine correct intubation of the trachea. Scott[1] states: 'The detection of breath sounds is notoriously misleading as these may be mistaken for the sounds of air passing along the oesophagus'. Our results support this. In six out of 40 ventilated oesophageal tubes, auscultation of the axillae revealed normal breath sounds. This confirms previous reports.[13,17,18]

The place of epigastric auscultation has also been emphasised[10,17] as has the combination of auscultation of the epigastrium and the axillae. We found this method in our investigation to be totally reliable whereas movement of the abdominal wall when ventilating an oesophageal tube was not a conclusive test.[5,17] Backflow into the oesophagus may occur from a stomach distended with air.[17]

We have confirmed that Wee's oesophageal detector device is reliable, as it was in the original paper,[9] whereas condensation of water vapour during expiration was totally unreliable as a test of tracheal intubation, as has been shown previously.[19]

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Recovery from anaesthesia in children

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Summary

There are no published comprehensive surveys of paediatric recovery room experience and the incidence of complications. A prospective survey was made of 16 700 consecutive admissions to the recovery room at the Royal Manchester Children's Hospital during the years 1985–1988. The incidence of respiratory complications was low, with laryngospasm 0.85%. The incidence of hypotension was higher than that in adult studies; over 50% of children recorded a decrease in blood pressure in the recovery room of more than 20%, compared to values before operation. The incidence of vomiting in the recovery room was also lower than in comparable adult studies. Certain aspects of recovery room practice changed during the 4 years of the study; these included routine oxygen administration, parents in the recovery room, and our approach to postoperative analgesia. The implications of these changes are discussed.

Key words

Paediatric anaesthesia.
Postoperative complications.

Previous reports on recovery room experience and problems associated with recovery from anaesthesia have been heavily biased towards an adult population.[1–3] There are no comparable series published for children, although Van Der Walt indicated in a study of 2260 children that 18% had at least one undefined complication in the recovery room.[4] The problems found in paediatric patients may be considerably different from those in adults. Furthermore, recovery room care is a developing area of paediatric nursing practice.

This paper reviews the work of the anaesthetic recovery room at a major paediatric centre, the Royal Manchester Children's Hospital (RMCH). The RMCH is a regional hospital of 191 beds with 91 beds for elective and emergency surgical admissions and five intensive care beds. We detail the complications during recovery and patterns of management for over 16 000 consecutive admissions to the recovery room after anaesthesia for a variety of surgical specialties and procedures during the years 1985 to 1988 inclusive.

Facilities and policies

The recovery room was opened in 1983 and has space for five recovery trolleys. Each bay has facilities for manual ventilation of the lungs, piped oxygen and suction, a sphygmomanometer and thermometer. In addition, one bay also has piped compressed air for mechanical ventilation. Monitoring equipment includes five ECGs, three automatic sphygmomanometers and three pulse oximeters (installed in 1988). In general, during the period of this study there was a decline in the use of invasive monitoring and this may reflect the increasing availability of noninvasive monitors. Resuscitation equipment includes two defibrillators and a fully equipped resuscitation trolley.

The room is staffed from 0900 to 2100 hours by three full-time and two part-time registered and enrolled nursing staff with no other duties, and is under the direction of a full-time recovery sister. From 2100 to 0900 hours the room is staffed by theatre staff and occasionally by recovery staff 'on call'. The sister in charge is responsible for inservice training, for trained nursing staff and student nurses on attachment to the theatre department.

It has been theatre policy since 1985 that all children who have general anaesthesia recover in the recovery room, apart from outpatient anaesthetics administered in the Oncology unit, which has separate recovery facilities. The routine procedures include: personal handover between the anaesthetist and the recovery staff, specific instructions on the anaesthetic chart, nursing on a one-to-one basis until the child has fully recovered according to our discharge criteria (Table 1), and a minimum of four sets of observations of pulse rate, blood pressure, respiratory rate and temperature are routinely recorded. Additional monitoring may be instituted by the anaesthetist or at the discretion of the recovery nurse; recently pulse oximetry was added as a mandatory recording.

All patients remain in the recovery room until discharged by an anaesthetist, but the vast majority stay a minimum of 30 minutes, in agreement with Steward's recommendations.[5] 1.19% of patients require admission to the intensive care unit. These are mainly planned admissions after

Table 1. Discharge criteria.

1. Able to maintain own airway.
2. Able to move all limbs.
3. Responds to verbal commands or speak.
4. Able to lift head for 4 seconds.

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Table 2. Demographic details of admissions to the recovery room.

	1985–6	1986–7	1987–8	1988–9	Total
Total cases	4137	3990	4012	4561	16 700
Cases during normal working hours	3544	3355	3408	3797	14 104
Cases after normal working hours	593	635	604	631	2463
Patients under one year of age	433	470	459	547	1909
Number of neonates (no preterm)	47 (12)	51 (11)	36 (10)	78 (31)	212 (64)
Number of patients in incubators	83	96	109	126	414

complex surgery or for continuing ventilation. However, this figure represents an underestimate of paediatric surgical patients who require intensive care, since some are transferred direct to the unit from theatre and therefore do not appear in the recovery statistics.

Patients

Between January 1985 and December 1988, 16 700 children were admitted to the recovery room. Of these 16 286 came directly from one of the three theatres, while 414 came from the X ray department after general anaesthesia for radiological procedures which included cardiac catheterisation. Surgical specialties represented at the RMCH include general, orthopaedic, otolaryngology, genito-urinary, oral, neurosurgery, cardiothoracic (except open hearts), and ophthalmology. RMCH is not the main neonatal surgical centre for the North Western region, although 212 neonatal surgical procedures were carried out there during the period of this review. Table 2 shows the number of cases per year in and outside normal hours, the number of patients under one year of age, the number of neonates and the number of infants who required incubator care in the recovery room. The number of neonates operated on at RMCH varied with bed availability at the regional Neonatal Surgical Unit. All neonates were nursed in an incubator or infant care centre, had an intravenous infusion *in situ* and were monitored fully with an apnoea alarm, intermittent measurement of the core temperature and inspired oxygen concentration.

The majority of theatre cases were performed during normal working hours, but there were also 2463 'after hours' emergencies during the study period (14.7% of the total). Recovery units do not always remain open for these cases,[2] but we consider they require postoperative recovery nursing and facilities of a standard equal to elective cases because of the occasional suboptimal pre-operative preparation and fasting necessitated by trauma and emergency surgery. Emergency anaesthesia is known to be a major risk factor in paediatric anaesthesia[6] and therefore, the unit is open 24 hours a day.

Parents in recovery

Parents are allowed into the RMCH's anaesthetic room during induction of anaesthesia,[7] and since March 1985 they have also accompanied their children to the recovery room. Their presence has increased yearly to our present figure of 69.2%. The criteria are that the child is awake and the airway and cardiovascular stability assured. Only one parent may visit, on request to the nurse in charge, provided that there are no problems with other patients and that the parent agrees to leave if asked. Guidelines are distributed to the parents before operation. Almost 70% of parents now visit the recovery area and no major problems were encountered during the study period. This figure would probably be higher if there were not occasional difficulties locating parents in the immediate postoperative period, especially when our predictions about operation length prove inaccurate. Ideally there should be a waiting area near the operating theatre suite for parents of children in theatre.

Complications

Respiratory complications. It is generally believed that the incidence of respiratory complication is higher in infants and small children than adults after anaesthesia.[8] However, in our series the incidence (Table 3) is low compared with other published series in adult patients.

Secretions in the oropharynx are common in the post-anaesthetic period and may cause laryngospasm.[9] 23.8% of patients in our study required oropharyngeal suction during their stay in the recovery room. This figure is high, possibly because of atropine usage for specific indications only, rather than as a routine. However, there were no major problems associated with the presence of these secretions in our series. 0.62% required suction as a result of blood in the oropharynx; the vast majority of these had undergone an ENT procedure. Our incidence of postoperative laryngospasm, 0.85%, is lower than previous reports in children of 1.74%.[10]

The low incidence of laryngospasm supports our policy of not routinely administering atropine to all children before anaesthesia. Laryngospasm in our experience usually settles with the administration of 100% oxygen together with assisted ventilation when necessary. Nineteen of our patients with laryngospasm required reintubation. Two of these had progressed to respiratory or cardiorespiratory arrest, but recovered fully.

Cardiovascular complications

There is a wide range of 'normal' heart rates and blood pressures in children of different ages.[11] Moreover, there are wide variations in atropine use (and more recently glyco-

Table 3. Respiratory complications.

Larygospasm	0.85%
Suction of secretions	23.80%
Suction of blood	0.62%
Controlled ventilation	0.16% (28 patients)

Table 4. Definitions of tachycardia and bradycardia according to age.

Age	Heart rate (per minute) Tachycardia	Bradycardia
<4 weeks	>180	<120
<1 year	>140	<100
>1 year	>120	<60

Table 5. Incidence of cardiovascular complications in recovery.

% decrease of systolic blood pressure related to value before operation	% patients
0–10	16.5
11–20	22.7
21–30	27.9
31–40	22.8
41–50	8.8
51–60	1.03
>60	0

pyrronium), intravenous fluid intake, choice of volatile agent, use of opiates and presence of postoperative pain which would influence pulse rates and blood pressure measured in the recovery room. Initially these values were not included in this study. However, we considered there was a high incidence of hypotension in the recovery room, and therefore this was studied prospectively during the last year of the study. Tachycardia and bradycardia were defined according to age (Table 4). Hypotension was graded according to the relationship between the lowest systolic blood pressure in recovery and the pre-operative blood pressure on the ward.

Table 5 shows that nearly all children showed a decrease in blood pressure in recovery; over 30% had a decrease in blood pressure of more than one third compared to the value before operation. This contrasts sharply with figures for hypotension of approximately 5% in adults.[12] This may reflect our liberal use of analgesia intra-operatively or may reflect differences in vascular tone between children and adults. The pre-operative blood pressure on the ward may be 'high' due to anxiety on admission to hospital.

The other problem with direct comparisons with adults is that we define hypotension as related to blood pressure before operation, whereas adult studies usually choose an arbitrary pressure such as 100 mmHg. Tachycardia (1.5%) and bradycardia (0.5%) after operation seem to be relatively uncommon in children, again possibly related to differences in definitions according to age.

Cardiorespiratory arrests

Ten children had a cardiorespiratory arrest. This was a primary respiratory arrest in five; the other five suffered cardiorespiratory arrest, but only one was a primary cardiac arrest. Details, complications, management, and outcome of these cases are shown in Tables 6 and 7. It can be seen that two cases of respiratory arrest followed administration of morphine, and two respiratory or cardiorespiratory arrests followed laryngospasm. One patient suffered cardiorespiratory arrest in the recovery room after rhinoplasty, possibly secondary to obstruction. (This patient was successfully resuscitated at the time, but died later from further complications.)

There was only one primary cardiac arrest, an incidence of less than 0.006%, and which was related to pre-existing pathology; the patient was successfully resuscitated at that time.

Fifty percent of our cardiorespiratory arrests followed comparatively minor, common ENT procedures which therefore require special vigilance during recovery. Seven of the 10 cardiorespiratory arrests occurred in the first 2 years of the study. We hope that continual review of policies and procedures and the increased use of pulse oximeters is improving our patients' welfare.

Nausea and vomiting

Nausea is entirely subjective and thus difficult to quantify especially in infants and small children. Incidence of vomiting, a more precise end-point, was 2.6% in the recovery period. Vomiting incidence in adult recovery units was reported as 4.5% in two series,[2,12] although Farman claimed an incidence of 2.5%.[1] Techniques of anaesthesia and choice of premedication obviously influence vomiting, but we consider our figures confirm the clinical impression that vomiting is less common after anaesthesia in children than in adults. This may be a true difference, or caused by differences in anaesthetic technique.

Table 6. Complications, primary respiratory arrests.

Age (years)	Sex	Weight (kg)	Operation	Complication	Management	Outcome
4	F	16.6	Adenotonsillectomy	Apnoea after intramuscular morphine postoperation	Intubation IPPV Intravenous naloxone	Full recovery. Discharged to ward after 2.5 hours in recovery room (RR)
3	M	13	Adenotonsillectomy	Laryngospasm Apnoea	Intubation IPPV	Full recovery. Discharged to ward after 3 hours in RR
7	F	21	Cystoscopy and cystogram	Vomit Apnoea ? aspiration	Intubation IPPV Chest X ray	Full recovery. Discharged to ward after 3.5 hours in RR
11	M	33	Posterior fossa exploration for astrocytoma	Progressive respiratory depression Apnoea	Intubation IPPV	Transfer to ICU. Permanent respiratory difficulty. Tracheostomy. Died from primary disease 15 months later
4	F	17	Adenotonsillectomy	Apnoea after intramuscular morphine given postoperation	Intubation IPPV	Full recovery. Discharged to ward after 3 hours in RR

Table 7. Cardiorespiratory arrests.

Age	Sex	Weight (kg)	Operation	Complication	Management	Outcome
5 years	M	20.4	Adenotonsillectomy	Laryngospasm Cardiorespiratory arrest	ECM Intubation IPPV	Full recovery. Discharged to ward after 2.5 hours in RR
5 weeks Born at 33 weeks	M	2.5	Pyloromyotomy	Cardiorespiratory arrest	Intubation IPPV	Full recovery. Discharged to ICU
16 years	F	39	Rhinoplasty	Cardiorespiratory arrest ? due obstruction	ECM Intubation IPPV Circulation and respiration restored within 5 minutes	To ICU for elective IPPV. Gradual deterioration. Died 6 days later
2 years	M	12	Laparotomy Disseminated lymphoma	Progressive respiratory difficulty ? cause	ECM Intubation IPPV	Died in RR
3 months	F	4.4	Cardiac catheterisation (VSD PDA Pulmonary hypertension Repaired oesophageal atresia)	Severe bradycardia Primary cardiac arrest	ECM Atropine Intubation IPPV	Full recovery. Returned to ward after 3 hours in RR

Changing patterns of recovery care

There have been major changes in certain aspects of recovery practice at the RMCH during this 5-year study, illustrated in Table 8.

Postoperative hypoxia in children in the recovery room is now well recognised.[13-15] This is usually unsuspected[13] and is not related to the conscious level of the child or the degree of recovery.[15] Colour change is an unreliable test for hypoxia since it depends on skin pigmentation, the site, and lighting.[16] There has therefore been an increased use of routine oxygen therapy, from 31 to 74.1% during the study period. This is thought to be responsible for the decrease in the need for oxygen to treat cyanosis, from 1.9% to 0.42%, and the recent availability of pulse oximeters has led to their routine use.

Postoperative analgesia in children has previously been neglected[17] and analgesia for babies is particularly controversial.[18] Attempts have been made at the RMCH to provide more satisfactory analgesia for these patients. There have been increases in the administration of opiate analgesics in recovery both intramuscularly and intravenously by infusion, in addition to a more liberal use of intra-operative analgesics. This has not led to an increased incidence of vomiting, but may have precipitated two cases of respiratory arrest after adenotonsillectomy. Surprisingly, the incidence of naloxone usage in recovery has decreased from 0.38% in 1985 to 0.25% in 1988.

Local analgesic techniques are increasingly popular, rising from virtually zero in 1985 to a current level of 11.9%. We have no specific policies for opiate analgesia in neonates and are cautious in its use because of the increased risk of respiratory complications.[19] Five neonates (2.35%) during our study period required intravenous naloxone to reverse respiratory depression.

Conclusions

Children have complications and problems in the immediate postanaesthetic period that are different from those of adults. Our complication rate was low compared to adult series. This may reflect true differences between children and adults after anaesthesia or may result from well trained staff, adequate monitoring facilities and standardised policies. It is to be hoped that other units will publish their paediatric recovery audits so that greater understanding of the particular problems and improvements in morbidity and mortality in paediatric recovery units can be achieved.

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Table 8. Changes in aspects of recovery room practice.

% Total cases	85–86	86–87	87–88	88–89
Oxygen for cyanosis	1.9%	1.17%	0.49%	0.42%
Routine oxygen	31.0%	48.2%	46.8%	74.1%
Intramuscular analgesia	3.4%	7.19%	16.3%	18.6%
Intravenous analgesia	1.66%	2.63%	2.84%	3.5%
Regional analgesia	—	6.1%	9.8%	11.9%
Parents in recovery	18.6%	46.9%	65.2%	69.2%

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Pain relief after tonsillectomy

Effect of benzydamine hydrochloride spray on postoperative pain relief after tonsillectomy

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Summary

The efficacy of benzydamine hydrochloride (Difflam) spray to relieve pain from postoperative tonsillectomy was assessed, but it was found that it did not relieve the symptoms after operation when compared to matching placebo.

Key words

Pain; postoperative.
Anaesthetic technique; topical.

Pain relief after tonsillectomy is often brushed aside as trivial. Patients suffer from dysphagia, otalgia, and inability to speak properly in the period after operation. The effect of benzydamine oral rinse has been investigated in the control of pain from pharyngitis and tonsillitis[1] and in patients who had tonsillectomy.[2,3] Raj and Wickam[3] included both children and adults in their study.

We studied the effect of benzydamine hydrochloride (0.15% w/v) (Difflam) spray to relieve postoperative pain and other symptoms in adult patients, because of their greater cooperation in answering a questionnaire about the effect of the spray. The study differed from the earlier ones in that it was a double-blind, controlled study and only adult patients (aged over 16 years) were admitted. Aspirin gargles[4] were not used because of their suggested antithrombotic effect.

The study was approved by the Ethics Committee.

Methods

Thirty-one patients between the ages of 17 and 32 years who were scheduled for tonsillectomy were included in this study. There were 13 males and 18 females. All of them were interviewed before operation and a written consent was then obtained. They were randomly allocated to receive either benzydamine hydrochloride or water as a placebo. Both the placebo and the active ingredient were supplied in 30-ml unmarked bottles. The placebo resembled the active preparation in all aspects, such as taste, smell and colour. Both of them were administered with the aid of a nurse using the Forrester spray.

The patients were premedicated with oral diazepam 10 mg, 2 hours before operation and had general anaesthesia that included thiopentone, suxamethonium and maintenance with nitrous oxide, oxygen and a volatile agent

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Table 1. Eight-hourly linear analogue scores for the placebo and the drug (active ingredient) group were ranked, and Wilcoxon's signed rank test applied, as shown below.

Time (hours)	Drug median $n=15$	Placebo median $n=14$	Wilcoxon's signed rank test significant at 5%
8	3.0	1.0	ns
16	6.0	5.0	ns
24	7.0	5.25	ns

ns, Not significant.

(either halothane, enflurance or isoflurane). All had tracheal intubation, were given 10 mg morphine intraoperatively and breathed spontaneously.

Patients were asked to assess their sore throat, ear ache and the state of swallowing, in the recovery room, just before return to the ward. Their symptoms were recorded on a linear analogue scale. During the postoperative period the tonsillar bed and the throat were sprayed by the nursing staff at least 3-hourly, up to four sprays each time. Observations were recorded three times a day for 24 hours. Paracetamol tablets or soluble aspirin were given if the patient complained of pain half-an-hour after the spray, and in some instances morphine was administered intravenously until they were comfortable.

Results

Out of 31 patients' results, 29 were suitable for the study. One was excluded because the solution was spilled accidentally, and one patient developed postoperative trismus temporarily and it was not possible to spray the throat properly. However, it was noted (when the code was disclosed later) that they were both scheduled to receive the placebo. Thus the placebo group was reduced to 14 while the group that received the active ingredient had 15 patients for analysis.

The results are shown in Table 1. There were no significant differences between the treated group and the placebo. Three patients in the treated group and four in the placebo did not require any analgesics. One patient complained of stinging and felt sick after the spray in the treated group, and one complained of stinging only, in the placebo group.

Discussion

Postoperative pain after tonsillectomy is thought to be the result of surgical dissection and the relative immobility of the pharynx, which causes muscular spasm in the pharyngeal muscles. Swallowing or attempted swallowing produce more pain. This deters the patient from swallowing which itself causes muscle spasm and further pain.

Benzydamine hydrochloride was introduced as a locally acting analgesic and anti-inflammatory agent. It is said it produces local analgesia by stabilisation of the cellular membrane and inhibition of prostaglandin synthesis. Hence it was considered preferable to the use of a local anaesthetic solution which might cause loss of pharyngeal and laryngeal reflex with consequent potential danger to the airway.

Difflam spray produced no significant pain relief in this series, although it has done so in acute pharyngitis, aphthous ulceration and mouth ulcers after radiotherapy. Young[2] studied the effects on children and showed that the analgesic effect was not significant the day of operation, but only on the first postoperative day. Our results show that Difflam does not relieve postoperative otalgia and sore throat in the first 24 hours.

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Prophylactic analgesia for daycase termination of pregnancy

A double-blind study with controlled release dihydrocodeine

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Summary

The incidence and severity of pain and nausea experienced by 40 primigravid day patients who presented for vaginal termination of pregnancy were examined. Controlled-release dihydrocodeine had no effect upon the incidence or severity of these minor

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sequelae. The requirements for escape analgesia and antiemetic therapy were less than anticipated and possible explanations are discussed. The low incidence of significant nausea and vomiting recorded in this study confirms that vaginal termination of pregnancy may be safely performed as day cases.

Key words

Analgesics, narcotic; dihydrocodeine.
Pain; postoperative.

The incidence of postoperative minor sequelae after gynaecological surgery is variable.[1,2] Patients who have vaginal termination of pregnancy (VTOP) have recorded the following side effects: nausea (25%), vomiting (17.5%), pain (20%), and drowsiness (60%).[3] Minimal emesis and effective pain management are important objectives within day surgical units. Newer techniques of analgesic medication administration may contribute to improvement of postoperative pain treatment.

The aims of this study were to examine the incidence of pain and nausea after vaginal termination of pregnancy in primigravid patients who have a standard general anaesthetic and to study the effects of controlled-release dihydrocodeine on the incidence of postoperative pain.

Method

Approval for the randomised, double-blind, single dose, parallel group study was granted by the local District Ethics Committee. Forty primiparous patients with pregnancies of up to 12 weeks' gestation, scheduled for daycase vaginal termination of pregnancy were recruited. Written informed consent was obtained and patients randomly allocated to receive either controlled-release dihydrocodeine 60 mg or a matched placebo tablet with 20 ml of water, one hour before surgery.

Anaesthesia was induced with alfentanil 7 μg/kg and propofol 2.5 mg/kg and maintained with a propofol infusion at 9 μg/kg/hour. Patients breathed a mixture of 70% nitrous oxide in oxygen via a Bain system, and arterial blood pressure, heart rate and ECG were monitored intraoperatively. One anaesthetist administered all the anaesthetics whilst another performed all the study assessments.

Patients were nursed in a recovery area in the immediate postoperative period and returned to the main ward area after 30 minutes.

Ten-centimetre visual analogue scales were used to assess pain and nausea; patients completed these at hourly intervals throughout their admission. Episodes of nausea and vomiting were recorded before discharge. Patients completed a questionnaire after discharge, to indicate if nausea and pain were absent, mild, moderate or severe upon arrival home, at their evening meal, in bed and on rising the next morning. Escape analgesia was 1 g paracetamol 4 hourly orally as requested. Details of all medication taken by the patients for 24 hours after surgery were recorded.

Results

The mean ages (SD) of both groups were 21 years (3) and duration of gestation (SD) was also identical at 9 weeks (3).

Nausea experienced in the daycase surgical unit is recorded in Table 1 and after discharge in Table 2. There were no statistically significant differences in the nausea experienced between the groups ($p > 0.05$ Kruskal–Wallace test).

Table 3 records the median, minimum and maximum visual analogue scores for pain assessments in the series, and Table 4 shows the pain experienced after discharge from the day unit. No statistically significant differences in

Table 1. Median, minimum and maximum visual analogue scores (mm) for nausea in the day unit.

Time	Dihydrocodeine group (n = 20)			Placebo group (n = 20)		
(hours)	Median	Minimum	Maximum	Median	Minimum	Maximum
0	0.4	0.0	1.8	0.7	0.0	6.6
1	0.0	0.0	3.1	0.0	0.0	5.6
2	0.0	0.0	8.3	0.0	0.0	3.7
3	0.0	0.0	1.8	0.0	0.0	4.0
4	0.0	0.0	0.9	0.0	0.0	1.0

Table 2. Nausea observed after discharge from the day surgical unit.

	Dihydrocodeine group (n = 20)			Placebo group (n = 20)		
Stage	None	Mild	Moderate	None	Mild	Moderate
Home	12	5	3	9	7	2
Meal	13	7	0	11	5	2
Bed	16	4	0	16	2	0
Morning	17	3	0	15	3	0

Table 3. Median, minimum and maximum visual analogue scores (mm) for pain after vaginal termination of pregnancy.

Time (hours)	Dihydrocodeine group (n = 20)			Placebo group (n = 20)		
	Median	Minimum	Maximum	Median	Minimum	Maximum
0	0.0	0.0	1.2	0.0	0.0	4.8
1	0.0	0.0	6.8	0.0	0.0	10.0
2	1.7	0.0	9.1	2.1	0.0	5.6
3	1.1	0.0	8.0	1.9	0.0	8.8
4	0.4	0.0	4.2	0.3	0.0	3.8

the results were recorded in Tables 4 or 5 ($p > 0.05$ Kruskal–Wallace test).

Three patients required atropine to correct bradycardia, a heart rate of less than 50 beats/minute. Six patients in the placebo and five in the treatment group received oxytocics. Eight received syntometrine and three were given syntocinon. Nine patients, four in the dihydrocodeine group and five in the placebo group required escape analgesia upon discharge from the unit.

Discussion

Over 150 000 terminations of pregnancy are performed annually in the United Kingdom and an increasing proportion are undertaken as day cases. There is little information available about the incidence of nausea and pain associated with this procedure in the primiparous patient, a group which we believe is prone to these complications.

There is no ideal general anaesthetic for termination of pregnancy. The policy in the Cambridge Day Surgical Unit has been to use propofol for induction and maintenance, supplemented by nitrous oxide in oxygen to avoid the myometrial relaxation associated with the use of volatile anaesthetic agents.[4] The use of oxytocics is avoided unless specifically requested by our gynaecologists. Only 5% of our series vomited and the incidence of nausea in both study groups did not present a problem. The visual analogue scores for nausea remained low within the day unit and the highest categorical scores were recorded on arrival home. Early mobilisation after surgery increases the incidence of nausea.[5] The low incidence of moderate and absence of severe nausea is even more surprising in that all patients received alfentanil 7 μg/kg at induction and 50% of the series also received dihydrocodeine. Propofol has been shown to have antiemetic properties[6] and this is further supported by our results.

The pain scores recorded were also low and no difference was recorded between the primigravidae who received dihydrocodeine and placebo tablets. The use of the visual analogue scale for pain estimation has certain limitations.[7,8] All patients were discharged between 3 and 4 hours after surgery and none were re-admitted.

This study was planned with the knowledge that 60% of primigravid patients had required simple escape analgesia in an immediate pre-study period. The use of prophylactic dihydrocodeine was expected to reduce the incidence of postoperative pain to 10%. The study was designed to detect a difference at the 5% alpha level with 80% power. However, the anaesthetic technique employed during this study yielded pain and nausea of insufficient severity to allow the proper assessment of the analgesic agents. A series of 600 patients would be required to discriminate between the need for escape analgesic therapy, assuming that the 28% and 20% figures for escape therapy in the placebo and actively treated groups were observed.

A marked reduction in the requirements for escape therapy was seen during the study although the anaesthetic and surgical techniques were unchanged from the immediate pre-study period. A number of factors may be responsible for this discrepancy. Thirty percent of the patients may be placebo reactors and a further reduction in the requirements for escape therapy may result from the apparent increase in concern shown to the patients by hourly visits from the medical staff during the research. The quality of the relationship developed between the patients and the assessor may have accounted for the high response rate (95%) seen in this study.

This study has demonstrated that a low incidence of nausea and vomiting may be achieved after anaesthesia for vaginal termination of pregnancy in primiparous patients. This procedure may be safely undertaken on a daycase basis with a high quality of recovery.

Acknowledgments

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Table 4. Postoperative pain after discharge from day unit. Number of patients

	Dihydrocodeine group (n = 20)				Placebo group (n = 20)			
	None	Mild	Moderate	Severe	None	Mild	Moderate	Severe
Home	9	10	1	0	8	8	1	1
Meal	15	4	1	0	9	9	0	0
Bed	16	2	2	0	16	2	0	0
Morning	18	1	1	0	17	1	0	0

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Alfentanil used to supplement propofol infusions for oesophagoscopy and bronchoscopy

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Summary

This randomised double-blinded study compared the cardiovascular stability and rate of recovery when propofol infusions with or without alfentanil were used to provide anaesthesia for rigid oesophagoscopy and (or) bronchoscopy. Forty-six patients were allocated randomly to receive either alfentanil 10 μg/kg or saline just before a rapid sequence induction with propofol. Suxamethonium 1 mg/kg was given and infusions of suxamethonium 10 mg/minute and propofol (10 mg/kg/hour for 10 minutes, 8 mg/kg/hour for 10 minutes and then 6 mg/kg/hour thereafter) were started. There were 23 patients in each group with no significant demographic differences between the groups. A significantly mean lower induction dose of propofol was needed in the alfentanil group (1.7 mg/kg compared to 2.2 mg/kg). Cardiovascular measurements were made on the ward pre-operatively, just before induction, just after induction, just after intubation, and at 3-minute intervals thereafter. Arterial pressure was significantly lower during the procedure in the patients who received alfentanil and there was a significant incidence of hypotension. There was no significant difference between the groups in respect of heart rate, with a significant increase in both groups just after intubation compared to the baseline values. Recovery from anaesthesia was assessed using the critical flicker fusion threshold. No differences were found between the groups and patients in both groups had returned to baseline values by 60 minutes. No patient had any recall of intra-operative events, and there were no other adverse effects of any significance.

Key words

Anaesthetics, intravenous; propofol.
Analgesics, narcotic; alfentanil.

Oesophagoscopy and bronchoscopy pose several anaesthetic problems. They are short but stimulating procedures, and the patients are often elderly with co-existing disease. Patients who require oesophagoscopy may have a significant risk of pulmonary aspiration of gastric contents, and a rapid sequence induction and rapid recovery of reflexes after anaesthesia are desirable. Ventilation of the lungs during bronchoscopy is commonly performed using an injector technique; inhalational agents cannot be delivered, and intravenous anaesthetic agents are used to maintain anaesthesia. There is a risk of awareness, especially if bolus doses are used, and prolonged administration of some intravenous agents may result in cumulation and delayed recovery. Combinations of an opioid with an intravenous anaesthetic agent have been shown to hasten recovery from anaesthesia[1] and to obtund cardiovascular responses to intubation.[2] Alfentanil and propofol have the shortest elimination half-lives of the opioids and intravenous anaesthetic agents available. This randomised double-blind study investigated the rapidity of recovery and cardiovascular stability when a propofol infusion with or without alfentanil was used to provide anaesthesia for rigid oesophagoscopy and for bronchoscopy.

Method

The study was approved by the District Ethics Committee. Forty-six patients who presented for rigid oesophagoscopy and (or) bronchoscopy gave informed consent to participate. No exclusions were made. The critical flicker fusion threshold (CFFT) was measured pre-operatively three times with an ascending frequency and three times with a descending frequency and the mean of these six readings recorded. The patients were allocated randomly to one of

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two groups; Group 1 received alfentanil 10 μg/kg just before induction of anaesthesia and Group 2 received saline. All patients were premedicated with temazepam 10 mg and metoclopramide 10 mg orally, 90 minutes before anaesthesia. An intravenous infusion was started on arrival in the operating room, and the patient breathed oxygen 100% until induction. Alfentanil or saline was given 3 minutes later, followed after one minute by an induction dose of propofol, given at a rate of 20 mg/10 seconds until loss of consciousness. Cricoid pressure was applied when consciousness was lost. Suxamethonium 1 mg/kg was given and infusions of propofol (10 mg/kg/hour for 10 minutes, 8 mg/kg/hour for 10 minutes and then 6 mg/kg/hour thereafter) and suxamethonium (10 mg/minute) were started. The trachea was intubated and the lungs ventilated with oxygen 40% in nitrogen during oesophagoscopy using a Bain system, with a fresh gas flow rate of 70 ml/kg/minute. Bronchoscopy was performed using a Negus bronchoscope with a Sanders injector. Cardiovascular measurements were made using a Critikon Dinamap 8466 with paper recorder at the following times: pre-operatively on the ward; in the operating room before induction; after induction but before intubation; 1 and 3 minutes after intubation; and at 3-minute intervals thereafter for 15 minutes. Hypotension and hypertension were defined as changes in systolic pressure of more than 33% of baseline. Hypotension was treated initially with intravenous fluids and if still present 3 minutes later by halving the rate of the propofol infusion until corrected. Hypertension was treated by a bolus of propofol 1 mg/kg. The propofol and suxamethonium infusions were continued until the end of the procedure. CFFT was determined in the Recovery Unit 30 and 60 minutes after anaesthesia, and the patients were questioned at 60 minutes about any memories or recall of the whole procedure. Statistical analysis was by ANOVA for repeated measures with pairwise comparisons using the Scheffe test, the *t*-test and the Chi-square test, with a significance level of 5%.

Results

The demographic and anaesthetic data are shown in Table 1. There were 23 patients in each group, and there were no statistically significant differences between these groups in respect of age, weight or duration of anaesthesia. Two patients in each group underwent bronchoscopy only, 15 in Group 1 and 16 in Group 2 had oesophagoscopy only, and the remainder underwent both oesophagoscopy and bronchoscopy. There was a statistically significant reduction of the mean propofol induction dose in the alfentanil group (1.7 mg/kg compared to 2.2 mg/kg).

Table 1. Demographic data; mean (SD).

	Age (years)	Weight (kg)	Duration of anaesthesia (minutes)	Induction dose (mg/kg)
Alfentanil–propofol	68.5	64.9	13.6	1.7*
$n = 23$	(14.4)	(13.4)	(5.7)	(0.5)
Saline–propofol	64.3	64.7	13.3	2.2*
$n = 23$	(10.7)	(12.4)	(5.6)	(0.6)

*$p < 0.05$ alfentanil–propofol group compared with saline–propofol group.

Figure 1 shows the changes in arterial pressure in each group during induction, intubation and the subsequent 15-minute period. There was a significant reduction in arterial pressure in the alfentanil–propofol group after induction compared to the baseline values. Patients who received alfentanil had significantly lower pressures compared to both the saline–propofol group and their pre-operative baseline 3 minutes after intubation and subsequently. The arterial pressure in the saline–propofol group was higher than baseline at all times but the difference was statistically significant only immediately after intubation. There was no significant difference in heart rate between the groups; in both groups there was a significant increase in rate just after intubation compared to baseline (Table 2). No arrhythmia was seen in any patient. The alfentanil–propofol group had significantly more episodes of hypotension; there was no difference in the number of episodes of hypertension (Table 3). There were no significant differences between the groups in rate of recovery of CFFT, and both groups had achieved baseline values by 60 minutes (Table 4). No patient had any recall of intra-operative events. Six patients had mild nausea (four in Group 1) and three patients vomited once in recovery (two in Group 1).

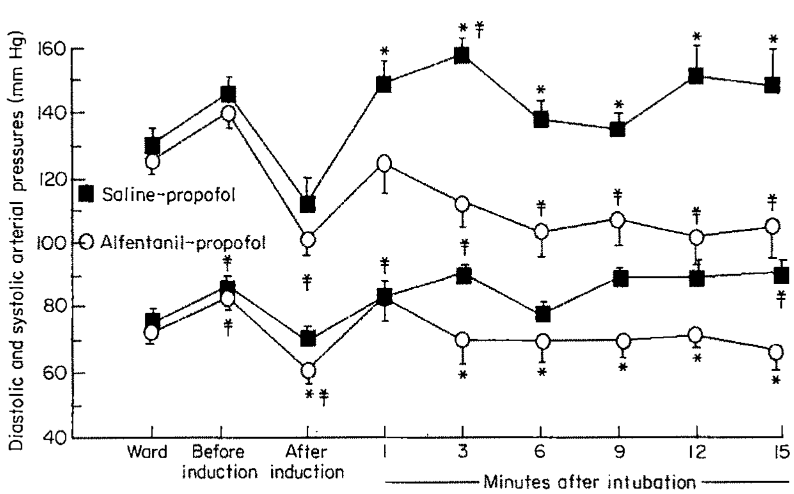

Fig. 1. Arterial pressure changes in the two groups. Bars indicate SEM. *$p < 0.05$ alfentanil–propofol group compared with saline–propofol group. ‡$p < 0.05$ compared with pre-operative baseline.

Table 2. Heart rate (beats/minute) during the study period; mean (SD).

	Ward	Before induction	After induction	Minutes after intubation 1	3	6	9	12	15
Alfentanil–propofol	76	79	76	88*	79	81	84	80	77
	(13)	(16)	(13)	(19)	(14)	(14)	(11)	(15)	(13)
Saline–propofol	74	79	81	93*	84	83	85	82	85
	(8)	(12)	(12)	(15)	(16)	(11)	(14)	(19)	(15)

*$p < 0.05$ compared to ward heart rate.

Table 3. Episodes of hypotension and hypertension in the two groups.

	Episodes of hypotension	Episodes of hypertension
Alfentanil–propofol	26	8
Saline–propofol	1	13
Significance	p = 0.0001	NS

Table 4. Critical flicker fusion threshold (Hz); mean (SD).

	Ward	30 minutes after operation	60 minutes after operation
Alfentanil–propofol	30.9	28.7*	30.1
	(2.6)	(3.2)	(2.7)
Saline–propofol	30.2	29.7	30.2
	(1.4)	(1.8)	(1.3)

*$p < 0.05$ compared to ward value.

Discussion

The propofol regimen was adapted from the study by Roberts *et al.*,[3] who showed that a loading dose of 1 mg/kg and a three-stage infusion technique achieved and maintained a stable blood concentration of 3 μg/ml within 2 minutes; this concentration provided satisfactory anaesthesia. Because of the need for a rapid-sequence induction in this study, it was decided to modify the scheme of Roberts *et al.* and to use an 'induction' dose of propofol in order to ensure unconsciousness during intubation. The study protocol specified that this induction dose should be greater than 1 mg/kg

It has been found that if intravenous anaesthetic agents are used to maintain anaesthesia, recovery from anaesthesia is faster if an opioid is given than if the intravenous anaesthetic agent is used alone.[1,4]. Recovery assessed by CFFT was similar in both groups in this study, despite the significant reduction in the induction dose of propofol after alfentanil pretreatment. However, the difference in induction dose between the groups is only 11.6% of the mean total dose of propofol in Group 2. Furthermore, the first CFFT measurement was made at a mean of 43 minutes after induction, and a substantial amount of the induction dose would have been metabolised by this time.

Alfentanil obtunded the cardiovascular responses to upper airway manipulation during the procedure, but there was a significant incidence of hypotension. These results are in accordance with the results of other studies of combinations of opioids and intravenous anaesthetic agents, including alfentanil and propofol.[2,5,6]

The conclusion is that both techniques can be used satisfactorily to provide anaesthesia for oesophagoscopy or bronchoscopy. The choice is made on clinical criteria for each patient, with reference to the different cardiovascular responses demonstrated in this study.

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Is nitrous oxide next?

It was with increasing anxiety that we read the latest Safety Action Bulletin from the Department of Health[1] which was critical of the use, and suggested the removal from anaesthetic machines of, carbon dioxide.

We agree wholeheartedly that carbon dioxide is a potentially dangerous gas, has been associated with anaesthetic morbidity and mortality and should only be used under strictly controlled and monitored conditions. However, where will the Department of Health directives go next? Will they suggest the removal of nitrous oxide? Here is an anaesthetic gas that can be delivered in a hypoxic mixture, expands compliant spaces, increases pressure in noncompliant spaces and causes diffusional hypoxia.[2] It affects vitamin B_{12} and folate metabolism[3,4] and its use during pregnancy is still being assessed.[5–7] Nitrous oxide is an operating complex pollutant and shows an association with spontaneous abortions in exposed female personnel.[8,9] It is not ozone friendly, may be adding to the greenhouse effect and is also expensive.[10] It must be a prime example of an agent that should be removed.

And then what? All anaesthetic volatile agents have potential side effects. Will they be removed? What about oxygen? In high concentrations for prolonged periods of administration oxygen can cause pulmonary complications. Will anaesthetists be left with only air?

Obviously, the answer is the judicious use of all agents exercising proper care and attention at all times. Instead of suggesting the removal of a useful gas we consider that the Department of Health should remind all anaesthetists of the potential hazards of carbon dioxide, and emphasise the correct use of end-tidal carbon dioxide monitoring.

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Beta adrenergic blockade masks a malignant hyperthermia reaction

We have strongly advocated that a persistent unexplained tachycardia should be taken as an early warning of a malignant hyperthermic (MH) response, and should lead the anaesthetist to check other variables (core temperature, blood gases, serum potassium, creatine kinase). Nowadays, as ECG monitoring is mandatory, tachycardia is the most easily accessible early sign rather than other signs of metabolic stimulation, such as increased CO_2 production. However, in situations where normal physiological responses may be masked by either concurrent disease or drugs, the limitations of the monitoring equipment and the information it yields must be remembered.

Control of blood pressure and heart rate during ear surgery is commonplace and it is frequently achieved by β-adrenergic blockade. In such circumstances a tachycardia, reflecting a hypermetabolic state, may well be masked by the effect of the β blockade until much later in the course of an MH reaction. It may well be too late then for effective therapy to be instituted. In this type of case ECG monitoring for MH cannot be relied on alone and extra monitoring should be performed either of end-tidal CO_2 production or of body core temperature.

More awareness of MH by anaesthetists and better monitoring during anaesthesia in general has had a marked impact on the mortality from MH in the United Kingdom, which is currently around 2–3% compared with 100% in the early 1970s. It is important to maintain and even improve our vigilance since it could be argued that patients should rarely die from MH which is so easy to diagnose and treat.

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Intensive care in the United Kingdom: Report from the King's Fund Panel

This timely report (*Anaesthesia* 1989; **44:** 428–31) highlights the expense of intensive care and the lack of information about its cost effectiveness. There are various trials, already well advanced, which consider this particular problem. However I must take issue with the statement that 'The panel recommends that each unit should prepare a set of guidelines setting out the criteria for admission to the Unit to help doctors and other staff determine priorities for treatment'. This is indeed a laudable aim with the heterogeneity of patients and the many different illnesses necessitating admission to Intensive Care Units but such guidelines, rigidly applied by the inexperienced, may deny intensive care to some who may benefit from it. Furthermore, inappropriate application of such guidelines will prevent progress since they would tend to exclude groups who only have a limited chance of survival. As an example, 15 years ago I was told never to ventilate artificially the lungs of any patient with renal failure because 'they always

die'. Would such a guideline be acceptable today? Mortality in such conditions remains high but most ICUs regularly treat such patients and there are many survivors.

We have had an admission policy for many years to inform our junior doctors and other staff. Decisions of this magnitude which involve patients' lives and other important issues, particularly the high cost, should not be delegated to a doctor who has merely read a set of guidelines. Consultants and only consultants must make these decisions if intensive care is to be properly conducted within the United Kingdom. This may mean disturbance at night for every admission to the Intensive Care Unit but that is the price to be paid for a safe, efficient system.

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A reply

Dr Park takes issue with the recommendations that every intensive care unit in the United Kingdom should have a clinical policy in the form of written guidelines. He may have misinterpreted the panel's intention that such guidelines should address questions relating to admission, discharge and the use of various interventions in individual units in such a way as to maximise benefit and minimise inappropriate activity. If intensive care is given to all who might survive, however small the chance, the admission of such a patient is likely to deny access to another more likely to benefit. It is surely unethical and improper to duck this issue. Guidelines should address this and other problems; they are not rigid instructions, but should be used flexibly as one criterion on which to base decisions. They might, however, be worded particularly strongly to guard against the eventuality that Dr Park envisages, namely the making of difficult decisions about the care of critically ill patients by inexperienced junior staff rather than by their seniors. It was precisely to avoid such things that the panel make this recommendation.

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Clinical freedom, clinical behaviour, and anaesthesia for laparoscopy

Healy (*Anaesthesia* 1989; **44:** 353–4) might find less to criticise if there were more recognition and teaching emphasis within the specialty of the many human factors common to the causation of accidents generally and to those arising from the interaction of the anaesthetist with his (her) environment, machines, colleagues and patients.[1] Although it is well accepted that 'prevention is better than cure' too often when reading of 'preventable incidents' vigilance[2] or performance[3] can be regarded below par, rather than the technique selected being at fault. His comments in regard to anaesthesia for laparoscopy appear to be based on hearsay and opinion, rather than practical experience and literature research, and make his suggestion of working within 'rules defined by responsible authority' alarming.

The writer has used mask anaesthesia in 155 patients, who required laparoscopic tubal ligation or diagnosis of infertility, over 4 years. The largest were 102 kg, 106 kg, and 110.5 kg respectively. The longest anaesthetic time was 70 minutes but most were completed in 20–30 minutes. Premedication was with lorazepam. Intravenous metoclopramide 10 mg was given before induction with thiopentone and nitrous oxide and oxygen (4:2 litres/minute) with 2% halothane through a Humphrey ADE system. Intravenous pethidine 25 mg was given after several minutes and the halothane reduced to 1% for the remainder of the procedure. Intravenous pethidine 25 mg was administered if anaesthesia was inadequate or for analgesia at the end. The muscle tone of the anterior abdominal wall in a few patients made it more difficult for the surgeon to grab hold and lift it up prior to insertion of the trocar. One can easily concur with Scott's unwillingness[4] to believe that anaesthesia for laparoscopy need be much more complicated than that required for other minor gynaecological procedures. Certainly, in my experience, minilap tubal ligation requires a deeper level of anaesthesia.

Older anaesthetists may remember patients put so steeply head down as to necessitate placing them directly on the nonslip mattress, rather than a stretcher canvas, with the additional aids of shoulder rests and bandaging of the legs to the leg supports to prevent slippage. Nowadays a skilled surgeon may complete the procedure in 5 minutes;[5,6] 2 litres CO_2 introduced into a horizontal patient produces an intra-abdominal pressure possibly less than 1 kPa, and just sufficient Trendelenburg tilt (0–10°) utilised to enable performance of the procedure. Published reports vary in so many aspects: premedication, induction, ventilation, relaxant, inhalational agent, CO_2 insufflation rate, CO_2 volume (3–20 litres), intra-abdominal pressure (1–5 kPa), Trendelenburg tilt (10–50°) and lithotomy/modified lithotomy positions, that little may be relevant to the technique Healy questions.

Mask anaesthesia was successfully used in large (15000,[7] 5000[8] and 2000)[4] and small[5,6] series of gynaecological laparoscopies. Others too[7,9] do not consider routine tracheal intubation is mandatory but suggest that anaesthetists always have the proper equipment, are prepared to intubate in every case and observe constantly the airway and monitor ventilation. Intubation is recommended if the surgeon is inexperienced, the duration unpredictable, or an airway problem pre-exists;[7] if excess Trendelenburg tilt or CO_2 volume is required; and for obese patients, those with adhesions, cardiac or pulmonary disease, or with other pre-existing problems.[9] Natof[9] points out the technique of intubation, even in skilled hands, has its own morbidity. It may also be relevant that the barrier pressure is maintained or increased in response to an increase in the intra-abdominal pressure.[10]

The comparative or actual risks of both methods are apparently undefined. Different choices need not signify failure to keep abreast of our subject or lack of responsibility but do pose the question of what is necessary to validate use of a particular technique?

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It was appropriate to publish the article under Anaesthesia and The Law on 'The awareness of pain during anaesthesia' next to Professor Healy's letter about clinical freedom (*Anaesthesia* 1989; **44:** 353–4).

Professor Healy's only choice of anaesthetic technique for laparoscopy, tracheal intubation and intermittent positive pressure of the lungs might produce just such a situation, quite apart from other potential hazards, such as kinked or misplaced tracheal tubes.

We submit that it is just as safe to anaesthetise for laparoscopy without tracheal intubation, or intermittent positive pressure ventilation, and it is much easier to tell if the patient is asleep.

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Subcutaneous morphine in children: taking the sting out of postoperative analgesia

Subcutaneous administration of opioids by continuous infusion through an indwelling 'Butterfly' type needle has become widespread in the management of pain due to cancer.[1,2] However the subcutaneous, as opposed to intramuscular, route has never achieved much popularity for postoperative analgesia. Indeed, a computer search of the English literature revealed only three papers concerned with this route of administration.[3–5] An indwelling intramuscular cannula was used by Harmer *et al.* for comparison of analgesics delivered by a patient-controlled device,[6] but this technique introduces a much higher degree of complexity and is also unsuitable for children.

Nursing resistance to the administration of intramuscular injections to children and the obvious distress it causes the patients prompted us to investigate the use of an indwelling subcutaneous needle for the administration of the standard '4-hourly' opioid injection. We conducted a study of children who had major surgery and present the results here.

Ten consecutive children, who had elective abdominal or thoracic surgery, in whom we anticipated that multiple doses of analgesic would be required, were included in the study. A 23-G Wallace Y-Can (dead space 0.15 ml) was inserted into the subcutaneous region of the anterior abdominal wall at the end of the operation but before recovery from general anaesthesia. The needle was removed, leaving the cannula in place and its patency checked by the injection of 0.5 ml saline 0.9%. The insertion site was then covered with a transparent adhesive dressing, to allow inspection of the site during injection.

The prescribed dose of morphine sulphate (10 mg/ml) was injected when indicated clinically by a nurse through the cannula which was then flushed with saline 0.5 ml. Note was made at the time of administration of pain on injection, leakage of fluid and redness at the site.

Details of the individual patients are given in Table 1. Nurses found the technique excellent or good in all cases, both from their own and the patients' viewpoint. Pain relief was also judged by the nurses to be excellent or good in all cases. Pain on injection was recorded by the nurses as 'none' in 35 out of 60 administrations (58%) and 'slight' in the remaining 25. One older child who complained of some soreness on injection still opted for injection through the cannula when offered an intramuscular injection instead! There was no leakage recorded during any injection and slight redness was noted in two patients after at least six injections had been given. No sequelae were encountered after removal of the cannulae.

Table 1. Details of each patient. Assessment scale : 5 = excellent; 4 = good; 3 = fair; 2 = poor; 1 = very poor.

Operation	Age	Weight kg	Dose mg	Number of doses	Pain relief	Acceptability: Nurse	Acceptability: Patient
Duhamel	10 months	8.2	1.5	8	4	5	5
Thoracotomy	7 months	7.6	1	10	4	5	5
Ureteric reimplant	10 months	9.6	2	4	5	4	4
Caecocystoplasty	11 years	37	5	6	4	5	5
Duhamel	9 months	9.5	2	6	5	4	4
Nephrectomy	4 years	16	3	5	5	5	5
Duhamel	11 months	7.7	1.5	9	4	5	4
Closure of ileostomy	14 months	8.2	1.5	4	5	4	4
Laparotomy	10 months	6.1	1	3	4	5	5
Sigmoid resection	13 months	7.8	1.5	5	5	5	4

The injection of morphine via a small cannula inserted subcutaneously therefore appears to be a safe, effective and acceptable method of administration in children. It seems surprising to us that such a simple technique should not, as far as we know, already be in use elsewhere and we would be interested to hear from any readers who do use it. We decided to use 'Y-Cans' rather than 'Butterfly' needles which are normally used in cancer patients, because of the danger of the needle becoming dislodged or embedded in an inquisitive child. In no case did the cannula become blocked or require reinsertion.

Forty percent of injections were associated with some discomfort, but this was minor compared with the obvious pain caused by the standard intramuscular injection. The technique has gained rapid popularity with the nursing staff who now request it for all children, where appropriate. We believe it deserves wider adoption and a formal comparison with intramuscular morphine would be of value.

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Tracheal tube cuff pressure

The paper by Willis *et al.* (*Anaesthesia* 1988: **43:** 312–4) and their letter on Profile-cuffed tracheal tubes and the Cardiff Cuff Controller (*Anaesthesia* 1989: **44:** 524) were interesting. They found that no adult females intubated with an 8-mm Portex Blue Line tube with standard cuff required a cuff pressure greater than 4.0 kPa to eliminate leakage past the cuff (mean minimum cuff pressure (MCP) 1.2 kPa), whereas adult males intubated with a 9-mm tube required a MCP of 5.3 kPa and suggested that this is because the difference in tracheal size between sexes is greater than the 9-mm to 8-mm difference in the tracheal tube size.

There is another explanation for this difference. In a report in which 200 adult tracheas were dissected and cross-sectional tracheal shapes studied and the circumferences measured, a higher incidence (24%) of asymmetrical cross-sectional tracheal shapes was found in specimens obtained from male patients.[1] Sixteen percent of tracheas from male patients were triangular, whereas no triangular-shaped tracheas were found in female specimens. Thus there was a definite correlation between sex and the tracheal shapes studied. The mechanism of sealing the trachea varies with the type of cuff used. The standard cuff of the Portex Blue Line tube is a low-volume cuff; it produces a tracheal seal by expansion in all directions and distorts the trachea in a circular fashion. It is, therefore, not surprising that in adult male patients with asymmetrical and triangular shaped tracheas, higher mean minimum cuff pressure was required to produce a tracheal seal. If the cross-section of the human trachea were always circular the sealing cuff pressure with a low-volume cuff would be lower. Male tracheal circumference was found to be significantly greater than female. Mean male tracheal circumference was 68.75 mm (range 60–79), whereas mean female tracheal circumference was 57.65 mm (range 50–69).

The profile-cuffed tracheal tubes do, I agree, have advantages over the standard-cuffed tubes since not only are lower cuff pressures required to maintain a leak-free seal but also the pressure in profile cuffs is more representative of the pressure exerted on the tracheal mucosa. However, one must point out that certain dangers are associated with both overinflation and underinflation of low-pressure cuffs. Overinflation of such a cuff may lead to application of excessive pressure against the tracheal mucosa, which causes not only destruction of ciliated epithelium with subsequent mucosal ulceration and stenosis of the trachea, but also acute dilatation and rupture of the trachea. Underinflation of the cuff may allow eccentric positioning of the tube in the trachea and leads to frictional mucosal erosion from the tip of the tracheal tube. There is also a potential danger of aspiration along the folds of excessive cuff material. It is reported that resting intracuff pressures as low as 1 kPa do not necessarily protect against aspiration, particularly since these large-volume cuffs are prone to produce longitudinal foldings of cuff material, forming channels through which liquid may travel down the trachea.[2,3]

It has become possible since the introduction of large-volume cuffs to measure and regulate the pressure exerted by the cuff on the tracheal mucosa, and it is now generally accepted that the low-volume cuffs should be abandoned for prolonged use. The benefits of large-volume, low-pressure cuffs are great enough in my opinion to warrant their use for both short and long periods of intubation in operating theatres and intensive care units.[4]

It is a great pity that measurement of cuff pressure is rarely performed in British anaesthetic practice.[5] The authors have shown conclusively that the present method of inflation using a syringe leads to gross overinflation of tracheal tube cuffs. Perhaps it is more practical and prudent to obtain an optimal tracheal seal with large-volume cuffs by producing a preset intracuff pressure. Intracuff pressure kept at a constant preset level of 2.5–3 kPa prevents both gas leakage and aspiration and will not interfere with tracheal mucosal perfusion.[6]

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A reply

We thank Dr Mehta for his erudite and informative letter. We are grateful for the information regarding the difference in the shape of male and female tracheas. He confirms our supposition that the difference between male and female trachea sizes is greater than 1 mm.

We believe that overinflation of high-volume, low-pressure cuffs may be more hazardous than overinflation of low-volume, high-pressure cuffs. This is because the pressure in high-volume cuffs is the same as the pressure on the tracheal wall. The design of cuffs is important and high-volume cuffs with folds are undesirable; such folds are unlikely to form if the cuff diameter at residual volume is roughly the same as the tracheal diameter. We agree, however, that properly designed high-volume, low-pressure cuffs should now be more widely used in clinical practice, provided that the cuff pressures are monitored and controlled.

Ideally, the cuff should be inflated to a pressure that minimises tracheal damage and at the same time provides a secure seal to prevent tracheal aspiration. This can only be achieved by using well designed cuffs that are inflated to a *fixed* low pressure of 2.5–3 kPa as suggested by Dr Mehta. This is particularly important in long-term intubation of patients in the Intensive Care Unit.

We have clearly shown the potential for gross overinflation of Normal and Profile cuffs with air-filled syringes. This effect will be accentuated during anaesthesia due to diffusion of nitrous oxide into the cuff. We have also investigated (unpublished data) the decrease in cuff pressure that occurs over long periods in patients on ventilators in the intensive care unit. This increases the potential for aspiration. These undesirable changes in pressure can be eliminated by the use of the Cardiff Cuff Controller.

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Inadvertent infusion of parenteral nutrition via the innominate artery

A 67-year-old man was referred to Intensive Care at the Regional Cardiothoracic Centre with acute aortic incompetence. He had initially presented with an acute confusional state and renal failure. A diagnosis of subacute bacterial endocarditis was confirmed by echocardiography which showed large vegetations.

The patient was hypoxic on admission to Intensive Care, with fulminant pulmonary oedema, and anuric. It was decided that he required an emergency aortic valve replacement, and in view of his condition, pre-operative ventilation. A pulmonary artery catheter was placed via the right subclavian vein and after stabilisation he was transferred to theatre.

Surgery was technically difficult; so was weaning from bypass. Intra-aortic balloon counterpulsation was instituted via the right femoral artery and eventually it was possible to withdraw bypass using the balloon pump with adrenaline and noradrenaline support. The immediate postoperative course was complicated by a re-exploration for tamponade.

The patient remained very unstable over the next fortnight and required considerable support. Multiple lines were inserted for inotropic drugs, feeding and dialysis. He stabilised and we started to wean him from ventilation and a tracheostomy was performed. A new triple lumen line was at this stage again inserted via the right subclavian approach. The line was preflushed with heparinised saline and inserted using the Seldinger technique, apparently without difficulty.

A check X ray, although it was rotated was believed to show the line in an acceptable position and feeding was commenced with Vamin glucose, 20% dextrose and 20% Intralipid via volumetric pumps.

Three days later, when reviewing a routine chest X ray (Fig. 1) we became suspicious of the line position. One lumen was disconnected from its volumetric pump and immediately bled back briskly. Arterial catherisation was confirmed by a blood sample and by pressure measurement.

This case demonstrates how easy it is to catheterise an artery inadvertently in a patient who has had multiple central venous catheterisations, especially if cardiac output

Fig. 1.

is relatively low, and emphasises familiar precepts. The colour of the blood obtained at vessel puncture does not provide reliable confirmation of venous puncture. Chest X rays taken to confirm the position of the catheter may be misinterpreted if they are rotated. The height of the column of blood supported by the pressure should be checked, or if possible the line transduced after insertion of a preflushed line.

There were no sequelae of the arterial infusion of total parental nutrition and the patient survived.

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Avoidance of unintentional arterial cannulation

Anaesthetists must be able to gain access to the central circulation consistently and rapidly. The incidence of carotid artery puncture during internal jugular cannulation is approximately 4%. Diagnosis may be difficult in the patient with hypovolaemia, hypoxaemia or impaired left ventricular function. Residual local anaesthetic in the needle and syringe which are used at first may give aspirated blood a bright red colour, and further confuse the picture. Avoidance of arterial cannulation is crucial for patients with coagulopathies, planned heparinisation or anatomical inaccessibility of a site for direct pressure (subclavian artery). We describe a rapid, simple technique to verify venous catheter insertion.

A sterile Novex 'K53' stopcock/tubing extension (Pharmaseal) is attached to the 18-gauge needle or catheter before insertion of the J-wire and introducer sheath. The tubing will fill and empty by gravity when lowered and raised, if the vein is cannulated. We have not found it necessary, but the system can be transduced as recommended by Jobes *et al.*[1]

We have used this technique without complications for cannulation of the internal jugular, subclavian and femoral veins in both adult and child patients. It is an integral training tool in our residency programme.

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Bradycardia associated with vecuronium

Cases of bradycardia associated with vecuronium are reported.[1–6] The bradycardia usually occurred some time after the administration of vecuronium, possibly related to vagally stimulating manoeuvres. A recent paper by Cozanitis and Erkola showed no decrease in heart rate due to vecuronium in healthy females who had elective hysterectomy.[7] We report a case of bradycardia in a critically ill patient when the temporal relationship of the bradycardia to the administration of vecuronium seems clear, and vagal stimulation due to other causes unlikely.

A 22-year-old woman, 10 days post-partum, was admitted as an emergency. She was dyspnoeic, hypoxic and hypotensive. The history, clinical signs, ECG and a chest X ray were all consistent with a diagnosis of massive pulmonary embolism. She was *in extremis*. She was resuscitated with oxygen, colloid infusion and adrenaline, and her trachea intubated after etomidate and suxamethonium. Ventilation of the lungs was continued by hand, on 100% oxygen. Referral to a cardiac surgical unit for emergency embolectomy was rapidly arranged. Approximately 15 minutes later 10 mg vecuronium was administered as an intravenous bolus in a peripheral vein to facilitate ventilation during transfer. She was haemodynamically stable (pulse 150 beats/minute blood pressure 80/60 mmHg) and oxygen saturation was stable at 85% by pulse oximetry before the administration of the vecuronium. Within 25 seconds of the administration of the vecuronium the heart rate decreased to 50 beats/minute for approximately 30 seconds, then increased rapidly back to 150 beats/minute without treatment. Remarkably, oxygen saturation remained stable.

It is impossible to state conclusively the cause of the bradycardia under these clinical circumstances. We are certain that there was no residual suxamethonium in the intravenous cannula. There was no evidence of worsening hypoxia or further embolisation at the time and temporally the bradycardia was related to the administration of vecuronium. There may be good arguments in the critically ill patient for the current emphasis on the use of agents reputedly without autonomic effect. However, if this case does represent a vagomimetic effect of vecuronium it could have been detrimental to this patient who was already hypoxic and who had poor peripheral circulation. We caution against the use of vecuronium by bolus injection in these circumstances. Perhaps the use of an agent with moderate sympathomimetic effect would be more appropriate.

The patient underwent successful pulmonary embolectomy.

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Laryngeal obstruction in HIV infection

A 30-year-old man was admitted to hospital complaining of a hoarse voice, dry cough and difficulty in speaking of one week's duration. He was known to be HIV positive, having been diagnosed in 1986 when living in the United States of America. He had recently developed AIDS, became unable to look after himself and was brought to the United Kingdom to be cared for by relatives.

He was awake and alert. He was apyrexial and there was

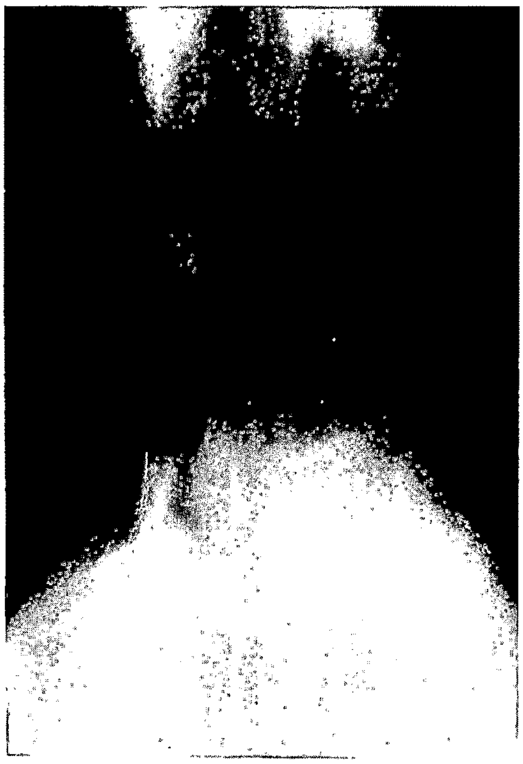

Fig. 1.

no lymphadenopathy or cyanosis. There was soft stridor, audible without a stethoscope; the respiratory rate was normal and there was no tracheal tug or rib recession visible. The chest was clear and air entry poor. He had difficulty speaking and did so with a high pitched voice.

Chest X ray revealed a grossly narrowed trachea, and laryngeal tomograms confirmed tracheal narrowing with a diameter of about 3 mm (Fig. 1). CT scan was also performed and showed a distorted narrowed trachea (Fig. 2).

The patient was obviously in imminent danger of tracheal obstruction and emergency tracheostomy was indicated. It was performed under local anaesthetic and was uneventful. The procedure was performed in theatre with an oxygen mask and secure intravenous line *in situ*. No sedation was given. A general anaesthetic was administered and direct laryngoscopy performed immediately after insertion of the tracheostomy. A distorted larynx was visible with a necrotic subglottic swelling which was biopsied. No tracheal lumen was seen. Subsequent histology showed the tumour to be a lymphoma of the nonHodgkins type and evidence of tuberculosis of the larynx as well.

Fig. 2.

Tracheal and laryngeal tumours are more commonly associated with HIV infection than is realised.[1-4] The larynx is involved in approximately 20% of patients with Karposi's sarcoma. What is rare, however, is for obstruction of the larynx to occur.[2] This patient presented with a very severe degree of laryngeal narrowing. We believe intubation of the trachea would have been impossible and awake emergency tracheostomy was clearly the only way to proceed.

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The incidence of awareness during anaesthesia

We read with interest the recent article by Pedersen and Johansen (*Anaesthesia* 1989; **44:** 504–8) about serious morbidity attributable to anaesthesia.

The authors reported that awareness had occurred in eight (0.1%) of 5926 patients who had undergone general anaesthesia. Awareness in anaesthesia is commonly assumed to take place in about 1% of all surgical patients,[1] so the reported incidence by Pedersen and Johansen seems rather low.

One of the reasons for this discrepancy may be their method of data collection: only patients who spontaneously reported that they were conscious during some part of their operation, were recognised to be aware. Many patients with recollections of intra-operative events, however, are known to have serious difficulties in discussing their experiences with the hospital staff for fear of disbelief or ridicule.[2] Blacher[3] found that the overwhelming stress of awakening whilst paralysed during surgery may cause postoperative traumatic reactions with amnesia for intra-operative events. It is therefore tempting to speculate about the two patients in the article by Pedersen and Johansen who had severe psychological sequelae after normal anaesthesia and surgery. These patients might have been suffering from latent psychiatric

disturbances, but they might also have been aware during surgery.

A recently published study by Flier et al.[4] reported two of 140 patients with recall of awareness during general anaesthesia. These investigators used a somewhat different approach (adapted from Brice et al.[5]) to assess the number of patients with intra-operative memories. All patients were asked during a short postoperative interview three questions: What is the last thing you remember before going to sleep for your operation? What is the first thing you remember on waking up after your operation? Do you remember anything in between?

A careful interview of patients, with direct questions about whether or not they have memories of intra-operative events, seems to be the appropriate strategy for assessing the incidence of awareness. If Pedersen and Johansen had used this method they would, without doubt, have found more patients with recollections of intra-operative events in their sample.

Pedersen and Johansen have done valuable work in pursuing types and frequencies of complications attributable to anaesthesia, but we suggest that in future studies on the incidence of awareness patients should be interviewed with scrutiny.

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A reply

We thank M. Jelicic and B. Bonke for their comment about awareness in anaesthesia, one of the serious complications found in our study, and in other studies as well. It may well be that we have missed a few cases during our postoperative interviews by not focusing specifically on this complication, using the form of interview suggested.

It must be borne in mind, however, that our material consisted of general anaesthesia as well as regional analgesia. The higher the incidence, the more disturbing is the situation. Awareness is an indication of faulty anaesthetic technique, and its frequency calls for renewed evaluation of some current methods, in order to protect our patients from this disturbing and sometimes morbid experience.

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Awareness with amnesia during total intravenous anaesthesia with propofol

A 30-year-old 65-kg teacher was scheduled for a 60-minute operation on her ankle. She gave an impression of appropriately positive attitude and had a composed and pleasant mood during the visit before operation. Nothing of importance was recorded either from the history or the records. It was decided to omit premedication. Induction was with a bolus of 150 mg propofol preceded by methyl-atropine 0.25 mg. Fentanyl (0.2 mg) was followed by 6 mg vecuronium whereupon the patient's trachea was intubated and the lungs ventilated with oxygen: end-tidal carbon dioxide was kept between 4 and 4.2 kPa. Propofol was infused at 10 mg/kg/hour and was kept at that rate from the start since the cardiovascular system was stable.

Nothing untoward was noticed until the 45th minute of the procedure. She then opened her eyes and moved her head. None of the monitored variables changed. She was instructed not to move her head in order not to cough. She closed and opened her eyes as requested and thus we obtained the information that she heard us and that she felt fine. She was told that she would be interviewed about this 'chat' postoperatively. An additional 2 mg vecuronium and a bolus of 50 mg propofol were administered. The operation was completed and the 10-minute recovery from anaesthesia was uneventful. The patient could not recollect the intra-operative conversation although she appeared quite awake, cheerful and orientated. She was amnesic for the first interview an hour later. A day later, she could remember only the later postanaesthetic interview and was surprised to learn that she had been conscious during anaesthesia.

A (possibly incomplete) review of the available literature about propofol revealed no case of conscious awareness during total intravenous anaesthesia. The producers of the drug are also unaware of a similar case, particularly since a high infusion rate was maintained. There are two possible explanations for this occurrence. Either the patient developed tolerance to the anaesthetic but not to the amnesic effect of propofol, or she metabolised the drug more quickly than is usual. The observed amnesia comes as no surprise. Patients often appear quite awake but remain amnesic for a considerable length of time after an anaesthetic with propofol.

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Visible expiratory valves

Shrouding of anaesthetic expiratory valves in order to allow scavenging of anaesthetic gases and vapours prevents the direct observation of the valve's action, but a modification of a Medishield valve described by A.J. Sansome and R. Bacon (*Anaesthesia* 1989; **44:** 425–7) restores this. There is also a practical application: a valve with similar features to the one described acts as a valuable aid not only for teaching but also in the pursuit of safe anaesthesia in, for

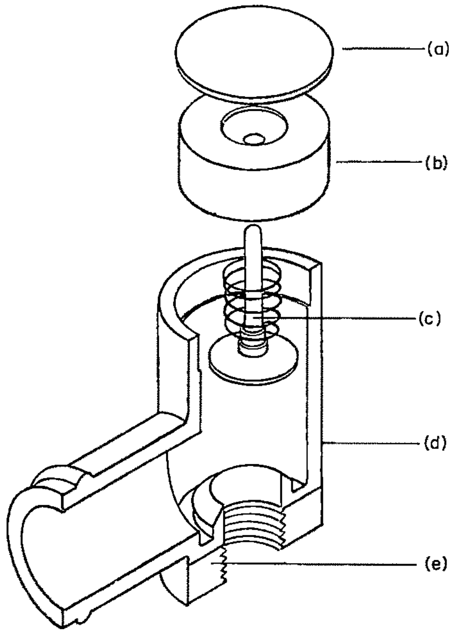

Fig. 1. The hybrid valve, which consists of (a) the lens; (b) new nylon valve guide; (c) the Ruben valve and spring; (d) the Penlon valve shell; (e) new threaded base in brass.

example, chairside dental anaesthesia. Loss of a view of the valve's movements is not just of academic interest since the manipulations of the surgeon in the patient's mouth are ever-present threats to the patency of the airway.

The popular McKesson flap valve was clearly visible in action. The chromium plated valve disc acted like a mirror and thus magnified its movements. This valve is no longer manufactured but the associated nosepiece and tubing assembly are still available and have the advantage of being self-retaining on the supine patient. Shrouding this valve was feasible if difficult but the inability to monitor the patient's breathing by watching the valve's action outweighed the benefits of scavenging.

When shrouded Heidbrink valves began to appear in hospital practice they were, and still are, larger and much heavier than their predecessors and are not really suitable for attachment to a dental nosepiece. However, the duct expiratory valve (Penlon) is transparent and the valve action is visible, as it is also in the Ruben nonrebreathing valve whose expiratory poppet valve is yellow. An ideal valve would combine the best features from all these three valves. The mechanism to adjust the pressure on the valve spring (which is unnecessary[1]) is omitted. When manual ventilation is needed a full facemask and a separate oxygen supply should be used.

The hybrid valve (Fig. 1) is constructed from: the transparent shell from a Penlon valve which contains the valve seat, the yellow poppet valve with its attached spring from a Ruben valve, and finally the movement of the valve is magnified by incorporation of a lens. This lens also serves to make the new valve assembly gas-tight by containing any slight leakage which might otherwise occur along the valve stem. Construction of the valve is much easier than the one described by Sansome and Bacon because the more intricate parts are already machined in the donor valves.

This valve has been used by the writer in over 8500 cases, mostly children, with passive scavenging. Its main virtue is to focus attention on the patient rather than on a remote reservoir bag or an electronic monitor. The high incidence of low oxygen saturation in children during general anaesthesia for exodontia suggests that greater vigilance is needed to detect respiratory complications.[2] This valve helps to increase vigilance and, in my own practice, the introduction of pulse oximetry has confirmed that the onset of hypoxia can, in most instances, be anticipated by observation of ventilation, including movements of the expiratory valve; corrective action can usually be taken before the oximeter alarm sounds.

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A reply

We agree with Dr Monro's comments on the value of restoring the information lost by shrouding.

The amount of valve movement that occurs with either the Heidbrink or McKesson valves is small and dependent on the flow through the valve. Our modification, by recessing the valve disc into a short sleeve, ensures that a clear indication of valve opening is provided. The valve disc must rise 3.5 mm before gas flow can occur. Thus, obstruction of the airway is differentiated from failure to obtain a seal. The hybrid valve described by Dr Monro cannot make this differentiation. Our modification does require some simple machining but should be within the capabilities of most departments of medical engineering. We chose to base it on one widely available expiratory valve to avoid using parts from several manufacturers, and to facilitate commercial production. The hybrid valve is constructed from components not originally intended to go together; we would be interested to see data on its performance.

It may be unnecessary to permit adjustment of the valve opening pressure in outpatient dental practice, but we think this facility is useful for teaching.

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Cyanosis in a parturient; central or peripheral?

One day this summer 3 hours after forceps delivery, a 40-year-old primagravida complained of headache and began shivering uncontrollably soon after arrival on the post-natal ward. The anaesthetist was summoned as she appeared noticeably cyanosed; however she was not dyspnoeic and denied any chest pain. She was pyrexial (38.3°C), blood pressure and pulse were normal but her tongue, face and hands were purple.

Epidural analgesia had been provided some 10 hours earlier, and had always been more effective on her left side. The patient reported its effects had largely disappeared, but when her feet were uncovered the left foot was distinctly

warm and indeed pink whereas the right was cold and blue.

Cyanosis is a notoriously imprecise clinical sign especially when the incident light is reflected, but there was no doubt in this case that cyanosis was present. It was considered at first to be central; despite no other symptoms or signs pulmonary embolism was feared; however, the residual effects of the epidural demonstrated that the cyanosis was not central in origin but peripheral.

Paracetamol was given for the headache, her symptoms abated over the next 20 minutes and were attributed to the change in ambient temperature on transfer from the stifling labour suite to the cooler post-natal ward.

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Pulse oximetry in the recovery room

We would like to report our own data of 968 patients supporting the usefulness of pulse oximetry in the post-anaesthetic recovery room (PAR) in relation to the report by Drs Smith, Cunning and Crul (*Anaesthesia* 1989; **44:** 345–8).

4.2% of our patients showed on admission an arterial saturation of oxygen (Sao_2) measured by a pulse oximeter (Nellcor N-10) below 85%; 15.4% of all patients had an Sao_2 below 90%; no additional oxygen was given at this time. These patients with a Sao_2 below 90% and (or) clinical signs of need for oxygen received 2–4 litres/minute supplemental oxygen by nasal cannula. A trial of withdrawal of oxygen therapy was started after 30 minutes with close monitoring of the Sao_2. If Sao_2 was less than 90%, administration of oxygen was restarted for 15 minutes followed by another trial period.

Oxygen therapy could be stopped after 30 minutes in 34.4% patients and in 60.4% after another 30 minutes. However, in 5.6% patients, even after one hour of supplemental oxygen Sao_2 remained below 90% or decreased below 90% on withdrawal of additional oxygen: all these patients had pre-existing respiratory diseases, mostly chronic obstructive airway disease, and were discharged from PAR despite their Sao_2 in good clinical condition.

Statistical analysis (ANOVA, $p < 0.05$) showed some patient risk factors (obesity, ASA-status, age) and operative events (duration of anaesthesia, amount of blood loss) to be associated with postoperative hypoxaemia in these inpatients. However, Sao_2 levels of less than 90% were found in patients without these risk factors. Therefore we suggest that all patients should be monitored with a pulse oximeter in the PAR.

Pulse oximetry enabled us to screen our postoperative patients for the risk of arterial hypoxaemia, to select the patients in need of oxygen therapy, to decide the duration of administration of oxygen and to assess the time of discharge from PAR.

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Respiratory arrest after a caudal injection of bupivacaine

There is a simpler explanation for the complication described by Lumb and Carli (*Anaesthesia,* 1989; **44:** 324–5) for respiratory arrest after a caudal injection of bupivacaine in a child.

Children have mechanisms by which blood pressure is maintained at normal levels, even in the presence of extensive sympathetic blockade. This observation has been made by all authors with extensive experience in spinal or epidural anaesthesia in this age group.[1-6] The fact that apnoea appeared suddenly, with little change in cardiovascular status, strongly indicates that a total spinal had occurred. It is easy to insert the caudal needle a little too deeply and to perforate the dura.[5]

Twenty milligrams of bupivacaine would last for one hour. The child was premedicated with papaveretum, also received halothane with nitrous oxide and his lungs were probably hyperventilated during the period he was paralysed. All of these factors would make it, as indicated by the authors, very difficult to evaluate any residual analgesia remaining from the subdural injection.

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Discerning rebreathing during anaesthesia

It was a pleasure to read the paper by Chan *et al.*,[1] who have clearly shown the fallacy of using the minimum inspired carbon dioxide concentrations as a means of discerning rebreathing in afferent reservoir (i.e. Mapleson A type) systems.

Utilisation of end-expired carbon dioxide concentrations is more correct. This involves sampling gas from the tracheal tube. However, this method is not the best to use in clinical practice, since it is deficient in a number of ways. Reliability of the method is dependent upon absolute measurements of carbon dioxide levels. No change in the pattern of carbon dioxide is discernible when the onset of rebreathing occurs and finally, since the increase in end-expired carbon dioxide only occurs when rebreathing is established, the onset of rebreathing is only discernible after the event.

My original proposition[2] of the best method, is to sample the nature of the gas which is eliminated from the system. This involves sampling gas from the exhaust limb of the system. It is well established that all afferent reservoir systems selectively eliminate alveolar gas. It stands to reason therefore, that as long as fresh gas, containing zero levels of carbon dioxide, can be measured in the eliminated gas, it is impossible for rebreathing to occur.

The carbon dioxide tracings (Fig. 1) illustrate the method. The tracings were obtained while applying an afferent reservoir system (Lack type system) to the lung model previously described.[3] Sampling took place near to the gas elimination site which is near to the patient attachment end of the system. The plotting of V_D/V_T fractions in relation to fresh gas flow reveals the onset of rebreathing below 6.0 litres/minute (Fig. 2). The zero carbon dioxide trace becomes shorter as the flow rate is reduced. Rebreathing is clearly discerned by an above-zero carbon dioxide trace at 5.5 litre/minute (Fig. 1). The change in the pattern of gas elimination allows for a more practical approach to the accurate regulation of fresh gas flow in clinical practice. A more detailed description and an entirely separate and thorough evaluation of the method is to be published in the *British Journal of Anaesthesia.*

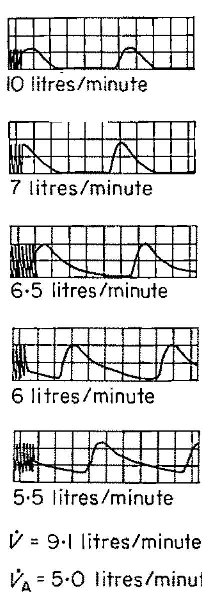

Fig. 1. Capnographic tracings obtained from sample site in the exhaust limb, 2 cm from the patient attachment end of the Lack system evaluated in Fig. 2.

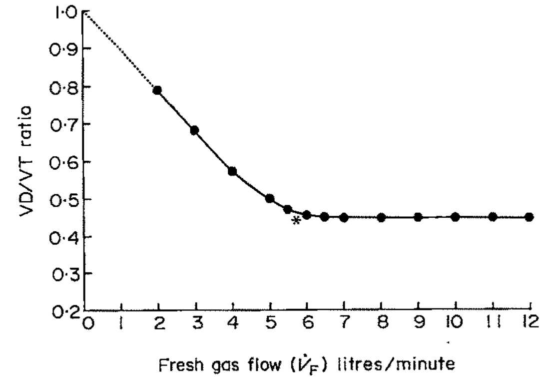

Fig. 2. V_D/V_T fraction in relation to fresh gas flow with a Lack type system, to ascertain the onset of rebreathing [*].

Chan *et al.* incorrectly attributed what they call the enclosed Magill system to T.J.V. Voss.[4] The first such system was designed and manufactured by me in 1980, and the design and results of an initial clinical evaluation were presented at an international meeting, the South African Society of Anaesthetists' Congress in April 1981. A provisional patent was filed in March 1981; the priority date for the final British patent was filed in March 1982. The reference[4] cited by Chan *et al.* does not describe the system. The first publication was in the British Patent office in March 1982, serial number 8208338, and to my knowledge the first description in a journal was in 1988.[5]

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A reply

The criticisms of the use of end-tidal carbon dioxide to assess the point at which rebreathing occurs, are accepted. What is required is a simple system which can be used clinically and in effect this requires a method that does not need capnography wave forms, since many of the commonly used capnographs do not have this facility. The system described by Miller appears to be ideal, at least for

afferent systems, if the value of zero during the ventilatory cycle can be used as described. This assumes that there is both selective venting of alveolar gas and that the amount of mixing which occurs within the expiratory limb is minimal. We look forward to reading the more detailed description, and in particular an evaluation of the method.

Readers should note that this enclosed Magill system was in use in Australia in 1980. It is obvious from Dr Miller's letter that the system was being developed in South Africa at the same time but we had neither knowledge of the system under development nor patented by South African designers until its appearance in the anaesthetic literature in 1988.[1]

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Desmopressin and bleeding

We read with interest the letter from Drs Spargo and Manners (*Anaesthesia* 1989; **44:** 363) which outlined the virtues and use of desmopressin acetate (DDAVP) to reduce bleeding after open heart surgery. A recent report showing that the median blood use in the UK was 5 units per patient suggests that there is considerable scope for improvement in blood conservation techniques.[1]

We agree with most of the introductory remarks of Spargo and Manners, but we wish to redress some of the bias in the latter half of their comments.

On the subject of efficacy, the study of Salzman *et al.*[2] did indeed show a reduction of mean (SD) operative and early postoperative drainage volumes of 40% from 2210 (1415) ml to 1317 (487) ml. There was a reduction in bank blood use from 3.7 units to 2.6 units per patient. These results have not been confirmed in further randomised double blind studies.[3]

The authors also implied that Salzman's study showed that a low pre-operative concentration of immunoreactive factor VIII-vWF was associated with a greater degree of chest drainage. These data were not statistically significant and the observation was described by the authors as follows 'an unexpected finding was a possible relation of vWF to subsequent blood loss.' It is difficult to interpret these data since all pre-operative levels were above the normal values in healthy subjects.[4,5]

This and other studies of pharmacological interventions aimed at the reduction of blood loss after cardiac surgery are the subject of a recent review.[6] One compound that was highlighted by your correspondents was aprotinin. Unlike DDAVP, the original observation of the beneficial effects of aprotinin[7] were confirmed in two further randomised double-blind placebo-controlled studies.[8,9] These results all show significant reduction in donor blood use with mean use of 0.3 and 0.5 units per patient. Eighty and 58% of treated patients respectively did not require donor blood at any stage.

The cost comparison between DDAVP and aprotinin is based on the current price for the formulation currently available in hospital pharmacies. This contains 100 000 KIU aprotinin in 10 ml of solution as well as benzyl alcohol, the preservative. This is not licensed for use in cardiac surgery or at the higher dose. It may prove toxic and give pseudo-allergic reactions if given in the higher dose regimen. The preservative-free preparation used in these studies is not available in the UK commercially. It is provided by Bayer UK Ltd on a named patient basis only. Cost comparison of a putatively beneficial therapy with a proven beneficial therapy seem at this stage to suggest more prejudice than insight.

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Buprenorphine as a premedicant in phaeochromocytoma

The necessity for adequate pre-operative preparation of patients with phaeochromocytoma is well known. Depth of anaesthesia is one of the most important factors in the prevention of hypertensive responses during manipulation of the tumour.[1] Narcotics as premedicants in phaeochromocytoma are suspected of inciting tumour activity. We used buprenorphine as premedicant in a case of phaeochromocytoma successfully.

An 18-year-old, 33-kg woman was admitted with persistent headache for 6 months, two episodes of convulsions with partial loss of vision for four months and a blood pressure of 210/140 mmHg. Her 24-hour urinary VMA and free catecholamines were 13.5 mg and 166 μg respectively. Diagnosis of phaeochromocytoma was made by ultrasonography and nuclear magnetic resonance. The patient was treated for 3 weeks before operation with labetalol 150 mg twice daily and captopril 12.5 mg twice daily. Her blood pressure was controlled at 120/80 mmHg (supine) and 90/70 mmHg (sitting). Both the drugs were continued upto the morning of operation. The patient was premedicated with 0.3 mg buprenorphine intramuscularly 1 hour before operation. She came to the operating room with a blood pressure of 128/80 mmHg, pulse rate 80/minute and CVP 12 cm H_2O. Anaesthesia was induced with thiopentone (5 mg/kg) and pancuronium (4 mg) was used for tracheal intubation and muscle relaxation. Her lungs were ventilated with O_2 and N_2O (66%). The blood pressure increased to 140/90 mmHg and her pulse rate was 96/minute during intubation; both remained stable throughout surgery. Her blood pressure was maximum at 157/110 mmHg during tumour squeeze and was minimum at 88/58 mmHg after ligation of the adrenal vein. There was no tachycardia. No vasodilators or vasopressors were used. The CVP was maintained at 13–15 cm H_2O. There were no changes at extubation. The patient was pain free in the immediate period after operation.

Buprenorphine reduces the heart rate by about 16% and systolic blood pressure by about 5–10%; its duration of action is 6 hours.[2] It is 30–40 times more potent as an analgesic than morphine. Buprenorphine 0.3 mg suppresses the cardiovascular and other clinical responses to surgical stimulation sufficiently[3] and is more effective than 0.125 mg fentanyl in the suppression of cardiovascular responses to surgery.[4] The anxiolytic and sedative properties make it suitable for pre-operative medication. It does not seem to cause histamine release. Buprenorphine could therefore be useful in patients with unstable circulation and this case report illustrates its use in patients for removal of phaeochromocytoma.

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Morbidity after use of the Finapres blood pressure monitor

The Finapres is a continuous noninvasive blood pressure monitor which displays an arterial pressure wave form and beat-to-beat variability. It operates using a photoplethysmographic technique, involving the placement of a small cuff around a finger. Ideally this is the middle phalanx of the middle finger. Volume and pressure changes under the cuff due to pulsatile blood flow are sensed, and cuff pressure is increased to restore the original conditions. The external pressure, continuously adjusted by the cuff, closely follows intra-arterial pressure in the digital artery, and allows measurement of the former as a function of arterial blood pressure.

Concern has been expressed about causing damage to the finger when using this device, but the manufacturer's guide states that continuous use for periods of 8–10 hours has caused no problems. Smith *et al.*[1] have reported no complications following continuous use for 7.5 hours, and Wesseling *et al.*[2] state it has been in use for 12 hours with no morbidity. However, Gravenstein *et al.*[3] noted discomfort and numbness in the finger after one hour. These symptoms were relieved by 30-second rest periods every 5 minutes. Analysis of capillary blood gas samples showed a deterioration even using this technique, when compared to continuous use. Thus the exact safe period for continuous use of the Finapres is not certain.

A 60-year-old man was scheduled for an estimated 5–6-hour operation for excision of carcinoma of the ear. This had spread to involve the inner ear and surrounding bone. He was otherwise in reasonable health, had no circulatory problems and his nutritional status was good; he was taking no medication. Surgery was extensively prolonged (17 hours) and involved three changes of anaesthetic nursing staff. The course of anaesthesia itself was stable and uneventful, and after delayed extubation on the Intensive Care Unit, the patient made a good recovery.

However, on the second postoperative day there was a large fluid filled swelling where the Finapres cuff had been placed. It involved the whole middle phalanx on its palmar surface, with several much smaller blisters on the dorsal surface. The finger was mildly painful, with flexion reduced by the bulk of the lesion, but sensation was unimpaired. Conservative management was successful and no sensory, vascular or motor loss resulted, and stay in hospital was not prolonged.

It is interesting that the problem was caused by direct cuff pressure, and did not involve the finger tip due to vascular stasis or occlusion. This seems to indicate the blood flow, although restricted, does continue through the finger, but that flow in the small vessels under the cuff may cease.[1]

The manufacturers are now reconsidering the wording of their guidance, but the safest course seems to be to limit the continuous use of the Finapres to 5–6 hours. Further use could be achieved by transferring the cuff to another finger.

It is also worth stating that if it is necessary for anaesthetic staff to change during a case, they should not only be aware of the course of the surgery and anaesthesia, but also be completely familiar with the monitoring apparatus, and appreciative of its limitations.

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Fluid flow through dural puncture sites

We read the paper by Cruickshank and Hopkinson[1] with interest. We too have performed a similar study of flow through cadaveric dura which we would like to present for comparison, because it differed in some aspects.

Samples of lumbar dura were obtained from the pathology department of the Freeman Hospital in Newcastle. A segment of the dura was mounted across the cut body of a 2 ml Becton Dickinson syringe, and a waterproof seal obtained using cyanoacrylate glue and adapted small cable hose clips. One sample of dura could yield up to 10 segments. The dura was kept moist at all times. The chamber of the syringe was then filled with oxygenated physiological saline and the preparation immersed in a water bath that also contained physiological saline at 35–37°C. The syringe was connected to a reservoir of saline via a Travenol blood-giving set at a pressure of 3.0 kPa measured using a manometer between the reservoir and specimen. The relatively high pressure was used in order to simulate the loss of fluid in the upright position, when headache is usually worse.

The dura was then punctured using either a 22- or 25-gauge Quincke-type spinal needle and the flow measured by counting the number of drops per unit time in the chamber of the giving set. A total of 10 samples were tested, five for each size of needle, with half the specimens from each sample being used for each bevel orientation. The flow was measured immediately after puncture, at 20 minutes, 24 hours and 48 hours after puncture in order to try and observe the loss of fluid over the time period at which headache usually occurs.

The results for puncture sites remaining patent at testing are shown in Table 1. Our results after initial puncture and 20 minutes later were similar to those found by Cruickshank and Hopkinson, and confirm the importance of needle size and the relative unimportance of needle bevel orientation in the initial period. There was also little difference in flows between the two orientations after 24 and 48 hours, although slightly fewer punctures were patent after puncture with the bevel parallel to the fibres. However, this was not found to be statistically significant.

Table 1.

Needle gauge and orientation	Percentage of punctures still patent			
22 parallel	100	97	15	9
22 horizontal	100	95	24	12
25 parallel	100	86	10	2
25 horizontal	100	90	15	5
	0	20 minutes	24 hours	48 hours

The lack of difference between the orientations is surprising in view of the strong clinical evidence in the difference in incidence of headache. Mihic[2] showed the effect of altering the needle bevel orientation to be of greater importance than needle size, with a reduction in the incidence of headache from 17.24% to 0.71% with 22-gauge needles, and from 15.15% to 0% with 25-gauge needles. We can only conclude that either loss of CSF is not the prime cause of post lumbar puncture headache, or that this experimental model does not reflect the *in vivo* state at all.

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Weight-determined dosage of atracurium besylate

Drs Harrison and Gunn (*Anaesthesia* 1989; **44:** 692) have suggested that doses of muscle relaxants should be determined by the fat-free mass rather than gross body weight from their experience with vecuronium bromide.

We found that the volume of distribution at steady state (VdSS) and clearance of atracurium were less in morbidly obese subjects.[1] All patients received atracurium on a mg/kg basis as determined by the gross body weight. The onset of neuromuscular blockade was faster, but its duration was not significantly longer in the obese individuals. The plasma concentrations corresponding to 50% recovery (Cp50) were 52% greater in the obese patients.

Neuromuscular relaxants such as atracurium are ionised compounds whose volume of distribution approximates to extracellular fluid volume (ECF). The smaller ECF volume per kg body weight in obese patients probably accounts for their smaller volume of distribution. However, obese individuals in our study required a higher plasma concentration of atracurium to attain the same degree of neuromuscular blockade as in lean individuals. This effect would counteract the alteration produced by changes in volume of distribution. We therefore concluded that obese individuals would require comparable doses of atracurium, on a mg/kg basis as subjects with normal body weight. It

may hold true for atracurium in obese subjects, but it remains to be shown for the other relaxants, such as vecuronium bromide.

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Propofol and atracurium in familial periodic paralysis

This is a report of the successful use of propofol and atracurium for two operations on a patient with familial periodic paralysis. The 26-year-old woman was admitted in the first instance for bilateral antral lavage. She had suffered from many episodes of periodic paralysis since infancy, a few of which were very severe. Five other members of her family and one of her daughters also suffered from the condition. One of her sisters had once required controlled ventilation of the lungs after an operation. The serum potassium levels of this woman and the other members of her family were normal during attacks. She was perfectly well at the time of admission and the laboratory investigations were normal. She received diazepam 15 mg, orally as a premedicant. Propofol 2.5 mg/kg, atracurium besylate 0.5 mg/kg and morphine 3 mg were given intravenously. The trachea was intubated and the lungs ventilated; anaesthesia was maintained with nitrous oxide, oxygen and isoflurane. Muscle relaxation was reversed at the end of the operation with neostigmine, 2.5 mg and glycopyrronium 0.5 mg. She was transferred to the intensive therapy unit for observation. Recovery was uneventful.

Eight months later the woman was readmitted for septal reconstruction, turbinate reduction and bilateral radical antrostomies. She received the same anaesthetics and again recovery was uneventful.

Familial periodic paralysis is an hereditary condition thought to be transmitted by an autosomal dominant gene. It is characterised by recurrent attacks of muscle weakness or flaccid paralysis occasionally associated with abnormality of the serum potassium. The skeletal muscles are affected and the bulbar muscles spared. Attacks may be precipitated by emotional excitement, high carbohydrate intake, severe exertion, cold, infectious diseases and accidental or surgical trauma. Thiopentone and muscle relaxants are also implicated.

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Cardiac arrest during laparotomy

We read with great interest the letter written by Drs Doyle and Mark (*Anaesthesia* 1989; **44**: 448). We encountered a similar occurrence recently during a total abdominal hysterectomy performed on a generally healthy woman of 46 years. She showed signs of extreme anxiety before anaesthesia despite diazepam 10 mg orally. She received 100 μg fentanyl and 0.25 mg vecuronium for pre-induction priming. Tracheal intubation was performed without any difficulties after injection of 250 mg sodium thiopentone and 8 mg vecuronium. Maintenance was continued with 70% N_2O in oxygen. Monitoring was by means of ECG, noninvasive automatic blood pressure, capnometer, pulse oximeter, and nerve stimulator to the ulnar nerve. Tears were observed in the patient's eyes at the first incision, and therefore a further dose of 100μg fentanyl was given. Shortly afterwards, when the uterus was being clamped by the surgeon there was a sudden decrease in pulse rate, from 80 to 50 beats/minute. Atropine 1 mg was immediately injected, but the bradycardia persisted until asystole was observed. The pulse oximeter ceased to demonstrate activity at the same time. The operation was immediately suspended, and another 1 mg atropine was injected. There was no immediate improvement so closed cardiac massage was started together with manual artificial ventilation of the lungs with 100% oxygen. Simultaneously 1 mg adrenaline was administered intravenously. The first ECG signs appeared shortly afterwards, which rapidly developed into tachycardia of 120 beats/minute; the blood pressure was now 130/80 mmHg.

The patient stabilised so it was decided to continue with the operation and, until the end of the procedure, anaesthesia was maintained with 0.6% halothane, 50% N_2O in oxygen, with the addition of fentanyl and vecuronium as necessary. There were no further problems during the remainder of the surgery or in the postoperative period. No signs were found of brain or cardiac damage, and the patient made no mention of awareness during the anaesthesia.

There is a similarity in our case to that of Doyle and Mark. Our patient also underwent an abdominal surgical procedure, did not receive anticholinergic drugs before the vecuronium, and responded with cardiac arrest during strong, painful stimulation under light anaesthesia. Contrary to the case cited, we continued with the anaesthesia after the resuscitation.

The absence of further complications emphasises the benign nature of vagally mediated cardiac arrest.[1] The bradycardia appeared shortly after administration of fentanyl and probably indicates a pharmacological interaction between this drug and vecuronium.[2]

Despite all the innovations and improvements in pharmacology, and in the use of sophisticated monitoring devices, our conclusion from this case is that it is essential to continue to observe the basic principles of anaesthesia.

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Asystole on induction

Dr Ruiz's letter on this subject was interesting (*Anaesthesia* 1989; **44:** 698–9) and this is another explanation of the event which he describes. The statement that no vagotonic drugs were used before the occurrence of the arrythmia is not accurate. Induction was with etomidate followed by suxamethonium to facilitate tracheal intubation. It is well known that both single and (more commonly) repeated doses of suxamethonium can cause bradycardia and asystole in animal studies[1] and in clinical practice.[2] Succinyldicholine and its hydrolysed product succinylmonocholine are structurally similar to acetylcholine. Several mechanisms have been proposed to account for the cardiac effects of suxamethonium including a direct action on atrial and ventricular muscarinic and nicotinic receptors; stimulation of postganglionic parasympathetic fibres to autonomic effector cells; stimulation of preganglionic autonomic fibres to sympathetic and parasympathetic ganglion cells; and stimulation of vagal centres.[1]

It was shown[3] that intramuscular premedication with atropine does not protect against bradycardia after the first or second dose of suxamethonium, but intravenous anticholinergics protect against this reflex in 80 to 90% of patients.[4,5]

Perhaps the administration of intravenous atropine before induction of anaesthesia would have averted the bradycardia and asystole described by Dr Ruiz.

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Hazard of piggy-back intravenous infusion and a possible solution

The danger of 'piggy-backing' intravenous infusions was reported recently[1] and this is a report of a similar case and a possible solution.

A 23-old-old primiparous woman presented in labour and requested an epidural. The epidural was performed after insertion of an intravenous cannula and a preload with one litre of compound sodium lactate. An infusion of syntocinon to augment labour was started through the same cannula using a Cardiff connector. This device[2] has two ports and a single hub to connect to the cannula. One of the ports contains a valve which prevents reflux of one intravenous solution into another. The patient continued to receive a slow infusion of compound sodium lactate, but 30 minutes later developed moderate hypotension with a systolic blood pressure of 90 mmHg. The cannula was then noted to be blocked and running poorly and so it was flushed and a rapid infusion of 500 ml compound sodium lactate solution was given. The patient then developed severe abdominal pain and the fetal heart tracing showed a profound bradycardia. It was realised at this time that the compound sodium lactate had been attached incorrectly to the Cardiff connector, and was on the port without a valve; this allowed the syntocinon to flow up into the compound sodium lactate solution, so that the patient received a large bolus of syntocinon. The infusions were stopped but the fetal heart rate remained low so arrangements were made to perform an urgent Caesarean section. This was performed under general anaesthetic, and the patient was delivered of a healthy baby after an uneventful operation.

Whenever drugs and other fluids are infused through the same cannula there is a risk of cross contamination of one with another should the cannula become blocked. Correctly used, the Cardiff connector prevents this, but there is the potential for confusion. One alternative is to insert two cannulae or use a double lumen cannula. The former is painful for the patient, so we describe an inexpensive and flexible version of the latter.

A Wallace Y-can cannula is inserted into a peripheral vein for intravenous fluids and if an infusion of a drug is subsequently required a Wallace Piggy-back cannula can be inserted through the Y-can so that the tip lies in the vein proximal to, and well away from, the tip of the Y-can. In this way the two infusions are kept separate.

Flow rates were found to be adequate for most purposes. The risk of cross-contamination was examined by occlusion of the vein proximal to both cannulae and by looking for cross contamination between the cannulae during injection of contrast material into the cannula under X ray control. In one case the contrast was seen to be prevented from flowing down the arm to the 16-g cannula by venous valves in the forearm. In the second case, although contrast did spill as far as the 16-g cannula it did not enter the giving set. In both cases the fact that the vein was occluded was obvious from the presence of blood in the giving sets. Should either cannula become blocked within its lumen there would still be no risk of cross contamination because the lumina are separate.

This arrangement offers the advantage that it can be set up as a single infusion to begin with and then converted to a double lumen cannula at a later time should it be needed, thus saving the pain and expense of inserting two cannulae, or inserting a double lumen cannula of the conventional type initially.

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The use of methylene blue in parathyroid surgery

Our letter (*Anaesthesia* 1989; **44:** 529–30) questioned the use of methylene blue in parathyroid surgery, because of the possible production of pain after an infusion of the methylene blue dye.

Twelve patients have now undergone removal of their parathyroids. All had an infusion of 7.5 mg/kg methylene blue in 500 ml dextrose 5% before induction of anaesthesia, according to the regimen of Dudley.[1] None of the patients complained of any pain before surgery except mild discomfort along the site of the infusion.

The cause of the pain in our study may well have been the very high local concentrations that may have occurred in the cuffed, exsanguinated arm. It seems that these concentrations are not observed in patients who undergo the infusion, because their skin colour did not change to such a profound extent as in our original experiment, and it was these areas of discoloration that were associated with the pain.

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Interscapular pain during epidural anaesthesia

Interscapular pain during epidural analgesia is reported[1-3] particularly amongst parturients. It appears to be uncommon in other patients but we have had experience of two nonobstetric patients who developed similar pain after epidural anaesthesia.

The exact pathogenesis of this pain is not clearly understood but it is believed to be due to the injection of air into the epidural space during loss of resistance test. This air possibly travels up into the thoracic and cervical region where it exerts pressure on the sensory nerve fibres supplying the dura and the spinal cord and produces pain. Epidural air can also produce other complications. The characteristic Doppler sounds of venous air embolism were heard in 8 out of 17 patients.[4] Hypotension and ECG changes were seen in one patient but serious complications could occur were this air to enter the left side of the heart in a patient with a right to left shunt. A patent foramen ovale directly contributed to the demise of one patient and probably produced disability in another after systemic air embolism.[5] Thus the use of air in loss-of-resistance tests should be discouraged.

Interscapular pain also occurred after the use of solutions for the loss-of-resistance test.[1,2] This is possible because, after the needle has entered the epidural space and is detached from the needle, the subatmospheric pressure in the epidural space allows atmospheric air to be sucked in. The volume of air thus aspirated depends upon the degree of subatmospheric pressure. Therefore, the use of solutions instead of air for loss-of-resistance tests does not seem to prevent this complication.

Entry of air may be prevented into the epidural space if a rubber cap, with a central slit in it, is attached to the hub of the epidural needle. The syringe is fitted to the epidural needle through the slit. When the syringe is detached from the needle on entry to the epidural space, the slit automatically closes and thus prevents ingress of air. The epidural catheter could also be passed through this slit. A three-way tap, between the epidural needle and the syringe, would also be preventive.

These methods and use of solutions for a loss of resistance test, prevent entry of air into the epidural space.

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Laryngeal mask and trauma to uvula

Drs van Heerden and Kirrage (*Anaesthesia* 1989; **44:** 703) are correct, blind passage of the laryngeal mask (LM) is not without hazard. A patient recently had a traumatic experience with the LM after surgery for varicose veins. She complained of severe sore throat after operation and the whole uvula was severely bruised. The rest of the pharynx appeared normal. This could only be caused by undue force during awkward insertion of LM. Not infrequently, the floppy tip of the deflated LM is deflected backward and upward and thus becomes stuck on the pharyngeal wall where it has to negotiate an almost right angle bend before it reaches the hypopharynx. A laryngoscope should then be used to aid LM insertion and avoid trauma to pharynx. Uvular trauma has not been reported previously.

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A reply

The laryngeal mask (LM) was developed over a period of 8 years and since then I have experience of approximately 8500 cases, and evolved a technique for insertion which

reliably avoids the problems encountered by Dr Lee without resort to laryngoscopy.

The introductory video emphasises the dangers of improper use in general terms and the need to avoid forceful insertion, but it does not demonstrate this technique adequately. The following text is a summary.

The LM cuff must always be tightly deflated in the recommended fashion before insertion. This imparts maximum rigidity to the cuff tip. Lubricant should be applied to the posterior surface of the cuff just before insertion. This is essential to prevent the cuff tip rolling over on contact with the palate. The neck must be kept flexed and the head rotated backwards by continuous pressure with one hand pushing behind, and under, the head throughout the insertion procedure. This will ensure a considerably greater than 90° angle at the back of the tongue in normal subjects and is an essential part of the technique. The cuff tip is inserted very carefully into the mouth under direct vision, holding the tube near its junction with the mask for maximal control. The most crucial part of the technique now follows: the mask tip must be pressed upwards against the hard palate until its dorsal surface is seen to flatten out smoothly against the palate. This can be quite difficult where the palate is narrow, where a lateral approach may be helpful. The mask tip should never be advanced further into the mouth until there is visual evidence that the distal third lies flat against the palate. This pressure upwards against the palate must be continuous to retain the flattened position and the mask is then advanced until it is completely inside the oral cavity. The tip then starts to pass downwards onto the upper posterior pharyngeal wall and is guided by contact with the uvula.

The hand position is changed at this point; stretch the index finger upwards to hook over the outer end of the tube, to prevent the device springing out of position. The tube is pushed in with a quick downward movement, using the minimum of force, until definite resistance is felt as the mask tip locates against the triangular-shaped base of the hypopharynx. There is a characteristic feel to this manoeuvre which with experience assures the user of correct placement. Resist the temptation to press the tip home several times. Instead, watch the front of the neck during this final movement, when both the thyroid and cricoid cartilages will be seen to move forward slightly. Similarly, both cartilages should move forward to form a smooth oval swelling when the cuff is inflated. If the thyroid cartilage only moves forward and there is resistance to inflation of the cuff, this indicates malposition.

Users of the laryngeal mask should carefully follow the above technique to ensure reliable and atraumatic use of the device.

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Difficult intubation aided by the laryngeal mask airway

The Brain laryngeal mask airway is a newly introduced apparatus that is an alternative to tracheal intubation in many anaesthetic situations.[1–3] We have used it as an aid for intubation when it was impossible to visualise the larynx by conventional methods.

Our first case was a 36-year-old for surgical excision of an olfactory neuroblastoma who had severe ankylosing spondylitis, with a fused cervical spine in the flexed position. Previously, intubation of his trachea was achieved by an awake, retrograde method which the patient was most anxious not to have repeated.

Pre-oxygenation was followed by induction with propofol 100 mg. Mask ventilation was found to be easy with an oropharyngeal airway and further increments of propofol, fentanyl, and atracurium were given before laryngoscopy was attempted. No view of the epiglottis or larynx was obtained at all. A size 3 laryngeal mask was passed with some difficulty aided by digital advancement of the tip to overcome the acute angle of the tongue base. The position of the mask was checked using a fibreoptic nasendoscope, then a 60-cm gum elastic introducer was passed via the mask into the trachea, and the mask removed. A 7.0-mm latex armoured tracheal tube was railroaded over the introducer and a posterior pharyngeal pack inserted and the operation continued uneventfully. Measurement of end-tidal carbon dioxide confirmed the intubation within the trachea.

Our second case was a 78-year-old man for revision craniofacial resection. Radiotherapy had caused severe limitation of mouth opening, and prevented insertion of the laryngoscope; a blind nasal technique secured his airway previously.

A size 3 laryngeal mask was inserted through the corner of the mouth where the upper molar teeth were missing, and then manipulated to a midline position after pre-oxygenation and induction with propofol. Controlled ventilation of the lungs was possible, so after muscle relaxation a gum elastic introducer was passed and the laryngeal mask exchanged for a 7.5-mm tracheal tube.

Our cases have shown that the laryngeal mask can be a valuable aid in performing blind intubation without the need for expertise or expensive additional equipment.

We consider that this technique could be routinely practised on anaesthetised patients to develop confidence before a difficult intubation is attempted. A prospective study is in progress to assess this technique.

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Priapism and general anaesthesia

Recently the urology journals have shown interest in the medical management of priapism. Successful agents are alpha adrenoceptor agonists, for example metaraminol (1 mg), ephedrine (50 μg), phenylephrine, or dopamine, injected into the corpus cavernosum.[1-3]

We had a case of priapism under general anaesthesia that prevented the urologist from passing the cystoscope. Ten milligrams metaraminol was injected into the corpus by the surgeon. This solved the problem, and the operation was over in 5 minutes. However, the patient developed ventricular tachycardia, that approached a rate of 200 beats/minute, with an arterial blood pressure of 200/140 mmHg. This settled without treatment and the patient made a full recovery.

Two deaths have been reported from the use of metaraminol to treat priapism; both were attributed to arterial hypertension. The development of ventricular tachycardia was potentially serious in this case; the dose was about 10 times that recommended.

The use of intravenous terbutaline (0.25–0.5 mg) was advocated[4] and may be a safe alternative.

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Failure to inflate the cuff of a tracheal tube

We wish to report an unusual case of apparent tracheal tube cuff rupture. A 30-year-old, para 2+0, woman presented at 39 weeks' gestation with an antepartum haemorrhage. Measured blood loss was about 500 ml; she was not shocked. However, because of the risk of concealed haemorrhage it was decided to proceed with Caesarean section under general anaesthesia.

An 8.0-mm internal diameter Vygon oral tube was passed easily, the cuff was inflated and ventilation of the lungs started. A leak was noticed after cricoid pressure was released. Cricoid pressure was immediately reapplied. The pilot balloon was seen to be deflated, so more air was added but it rapidly deflated again. Intubation was easy so we decided to replace the tube. The cuff on the replacement tube was checked and then the tube replaced. However the pilot balloon was again seen to deflate. Cricoid pressure was maintained and the tube replaced for a third time. Yet again the pilot was seen to deflate!

Disbelief set in: another cause for tracheal cuff deflation was sought. A green anaesthetic swab is used in this hospital to identify cuff syringes and part of the swab had become trapped between the barrel and the plunger of the syringe and allowed a slow leak of air. One syringe is used to test the cuffs and another to inflate them once the tube is in the patient's trachea. A new syringe was used and the cuff inflated satisfactorily. The two previous tubes were examined and their cuffs were intact.

The syringe used has only one narrow sealing ring on the plunger. This narrow single ring not only makes it more likely that a swab may be trapped but also makes it difficult to see. Other syringes have double sealing rings and so long as the swab did not cross both rings the syringes remain airtight. The defect is also easier to see.

This event would not have occurred if some other means of identification were used. However, if a swab is used to identify syringes then those with a single sealing ring, such as the one described here, should not be used. Finally, the same syringe which inflates the cuff once the tube is in the patient's trachea should be the one used to test the cuff before use.

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Royal Alexander Hospital, Paisley — E. WATSON

It is common practice here to insert a swab into a 10-ml syringe in order to set it aside for inflation of the tracheal tube cuffs. We needed recently to change the tracheal tube of a patient twice because the cuffs failed to inflate.

Later, we found that a small portion of the swab was caught between the syringe wall and the plunger. This allowed a leak of air to escape from the syringe and the subsequent failure of the cuff to inflate.

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A burst sphygmomanometer cuff during intravenous regional anaesthesia

Obesity is a commonly encountered problem in anaesthesia; morbid obesity is defined as a weight over 50% of the ideal. We encountered a 23-year-old patient whose height was 185 cm and weight 195 kg (260% of his ideal weight)[1] who had fallen off his Moped and sustained a Colles' fracture. His previous history was unremarkable, but he became short of breath when walking, had a productive cough and a resting blood pressure of 170/100 mmHg.

General anaesthesia was thought to be inadvisable, because of potential complications[2] and a local nerve block too technically difficult, so we decided to perform a Bier's block. The usual arm and leg cuffs were tried but they were

unsuitable because the arm cuff was too short. His arm circumference was 45 cm and he would not tolerate the pressure in the leg cuff because it was only 10 cm in width. An outsize thigh blood pressure cuff 16 cm wide was tried and, when inflated up to the recommended 100 mmHg above systolic pressure, was acceptable.[3] A routine Bier's block was performed with 60 ml 0.5% prilocaine, which gave adequate analgesia. However, after 12 minutes and with manipulation of the arm in progress, the cuff burst! The patient suffered no untoward effects, the reduction of his fracture was satisfactory and he returned home the next day.

Prilocaine in doses needed to perform intravenous regional anaesthesia is safe even during inadvertent failure of the cuff.[3] This case demonstrates the necessity of suitable outsize equipment for use in the morbidity obese patient. The blood pressure cuff is not designed to take pressures of 270 mmHg for long periods, or even higher pressures during manipulation of the arm, and we would not advise its use during this procedure.

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Hyperbaric cinchocaine

The publication of the article by Roberts *et al.* (*Anaesthesia* 1989; **44:** 471–4), has prompted numerous enquiries from Australian anaesthetists about the availability of hyperbaric cinchocaine (Nupercaine Heavy). The article suggests that the agent has been withdrawn from both the United Kingdom and Australia.

We would like to clarify that Nupercaine Heavy is still available in Australia and that we have no plans to discontinue its production in the foreseeable future. Our current range of spinal anaesthetics in Australia is therefore Xylocaine 5% Heavy, Marcain 0.5% (plain), Marcain 0.5% Heavy, and Nupercaine 1:200 Heavy.

Astra Pharmaceuticals Ltd, PO Box 131, North Ryde, New South Wales 2113, Australia

R.G. Cummins
L.J. Lilley

Subarachnoid anaesthesia with 0.25% isotonic bupivacaine for Caesarian section

The letter from A.J. Wilson about his experiences in the use of 0.25% bupivacaine for subarachnoid block for Caesarean section was interesting. My experience was in the same establishment as the writer and included the enforced use of the weaker local anaesthetic solution. The same technique was used, as Dr Wilson described, with 0.25% bupivacaine or between 2.0–2.2 ml 0.5% plain bupivacaine in about 75 patients in each group (although not in any controlled trial). His enthusiasm for 0.25% bupivacaine is not shared by me. One patient required a general anaesthetic for an inadequate block in the 0.5% group but four required general anaesthesia in the 0.25% group, and 15 patients required supplementary analgesia in the latter group, compared with none in the 0.5% group. There was slightly higher incidence of hypotension which required fluids and (or) ephedrine in the 0.5% group; this was offset by its more predictable level of block and a greater degree of motor block making surgery easier. If I had the choice, 0.5% bupivacaine would win every time.

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H.M. Munro

Anaesthesia, 1989, Volume 44, pages 1018–1020

Book reviews

General anaesthesia

Edited by J.F. NUNN, J.E. UTTING AND B.R. BROWN. Pp. 1434. Butterworths, 1989. £110.

I was almost overwhelmed at the prospect of reviewing this book: even if I were to read a chapter a day, the task would take about 3 months. Nevertheless, I set out with enthusiasm and was particularly encouraged by the preface which states that, despite the multitude of international authors, standard English would be used. The introduction of a distinguished American into the editorial team however, does rob one of the opportunity to describe the new edition of the old GNU as 'A gnother GNU'!

The first section of the book covers what are usually called 'basic sciences'. There are some excellent chapters, but others are merely lectures which we have all heard before except, this time, references are added. The 'pre-operative period' is a very well balanced section which covers the major systems that can affect anaesthesia. It also clearly reveals how modern investigations can be of great assistance to anaesthetist and patient. It is a section that is well worth reading.

The 'process of anaesthesia' is something of a mixed bag with an excellent chapter on the depth of anaesthesia and awareness. The physics of anaesthetic equipment are reviewed. The associated fundamentals of fluid replacement, hazards and record keeping are also discussed. The 'complications of anaesthesia' are generally well presented. There is an outstanding chapter on the management of peri-operative arrhythmias which should be read by all anaesthetists. This section on 'special problems inherent in the patient and environment', covers a great deal of ground both literally and geographically. Each author was selected for personal expertise but, unfortunately, the space available does not allow some of them to do justice to their subject. 'Anaesthesia for particular operations and procedures' covers a very large area: there is understandably some repetition, particularly in reference to the classic beginnings of a history, clinical examination, investigations and checking of apparatus. This is the most disappointing section, however, because the limited space allowed to authors denies readers the benefits of their knowledge; most chapters are also the subjects of books.

The part on 'regional anaesthesia' is short, sharp and mainly to the point! However, for the anaesthetist who wishes to practise local techniques regularly, it is inadequate. The 'postoperative period' is reviewed and each contribution varies from the adequate to the well done. The reader seems to be hit by fact after fact and yet there seems to be no concept of the philosophy of continuing and simultaneous care.

The section devoted to 'intensive care' is enjoyable. It is a balanced section and will be a valuable aid to the anaesthetist who has some involvement in this clinical area. There is a sentence by Dr J.C. Stoddart, who is considered by many to be one of the founding fathers in British intensive therapy, which should be placed above the entrance of each unit in the country: 'Overtreatment or unnecessary treatment can be avoided if everyone concerned with the care of the severely ill, tempers therapeutic zeal with attention to humanitarian principles'. Chapters on 'resuscitation, the pain clinic' and the inevitable 'medicolegal aspects of anaesthesia' conclude the volume.

The book went with me on holiday to the Mediterranean so that the editor's deadline for this review could be met. Hercules also performed great labours to the east of the Balearic Islands and, after reading all 113 chapters, I feel I too have laboured hard! This book contains a vast amount of knowledge but it is not easy to read. It is in fact, several books rolled into one massive tome. Some would say that this type of book is no longer necessary because there are others which cover for instance, basic sciences, cardiopulmonary anaesthesia and intensive care. One could also argue that some chapters have been superseded by more up-to-date publications. This book will be of limited value in 2 to 3 years' time.

The last question is, would I buy this book for myself or for the department library? The answers are that I would not buy it myself and would be inclined to compare it with the two others currently available, before recommending its purchase by anyone.

R.S. VAUGHAN

Day care surgery, anaesthesia and management

Edited by E.G. BRADSHAW AND H.T. DAVENPORT. Pp. xv+191. Edward Arnold, 1989. £25.00.

This slim 19-author volume is the first reference work to address the ideals and practicalities of day stay surgery in a British context. It aims at those establishing a unit, with a secondary target of encouraging those already involved. The book achieves its primary function. The chapters are clearly detailed in the contents. In a conservative start the historical information, statistics and tables touch on the icons of day surgery traversed in a 1986 US work, *Major ambulatory surgery* by J.E. Davis. The reader is led from Glasgow, Scotland (1908) via Phoenix, Arizona, USA (1970) through floor plans and lessons learned by a variety of authors in the present day. Reluctant readers who have this book thrust on them by colleagues will not be

affronted by the style or content. The anaesthetic tyro will be appropriately perplexed by ambivalent attitudes to intubation. Enthusiasts will have to read through it all to get best value. The report of the Royal College of Surgeons 'Guidelines for day case surgery' is gently rebuffed at several points; sensible, hands-on management wins the day. The information on costs is best glossed over, since the book's preferred UK site for such a unit (the district general hospital) involuntarily gives hidden subsidies.

Nurses will find depth in the offerings from Exeter and Barnet: it is vital that one person is given the responsibility to make it all work. On a day-to-day basis that person is a nurse manager, backed up by an interested consultant from the medical staff, usually an anaesthetist (surgeons may be too manipulative!). Incorporating a programme within existing theatres rarely works as well as the semi-autonomous unit. Private practice rates a whole chapter and numerous other paragraphs. Administrators will find it worth their time to browse through the whole book.

Market forces crop up at several points and apply to state and private sectors. Several chapters discuss the proportion of cases suitable for day stay, and two hint at viability levels: e.g. 2000 procedures per year. None use an antipodean figure that 10% of the population require procedures each year, but they broadly agree that 25–40% of those should be day stay. Some authors suggest that 'after hours' uses for the building should be encouraged, one example is the thoughtful pain clinic contribution. They do not comment on increased wear and tear or loss of morale that may result when things get moved by the 'others'.

What is missing? Do not look here for lists of equipment needed, inappropriate encyclopaedic recipes for the art of smooth anaesthesia, or, more than one job description. It is an introduction. Surgeons will not find the most important advice of all: try simple procedures first, then once you, your team, and your patients have confidence in the system, evolve. There is a sense of enjoyment, flexibility and enthusiasm that comes through the whole book. Day stay unit patients receive quality care; small unit management is personal and rewarding. This book embodies that principle. The authors will be asked for more.

A.K. BACON

Technical manual of anesthesiology

Edited by J.E. HEAVNER, C. FLINDERS, D.J. MCMAHON, T. BRANIGAN AND J.M. BADGWELL. Pp. viii + 167. Raven press, 1989. $32.50.

The second author of this book is described as '3rd year Medical Student, formerly Anesthesia Technician', which perhaps explains both the motivation for, and relative success in, producing a readable introduction to the technical equipment and background of modern anaesthetic practice. It is written uncompromisingly for the United States but restriction to transatlantic standards and legal requirements is not much of a drawback and, in some areas such as temperature monitoring, we can always hope that awkward questions from trainee anaesthetic assistants may stimulate improvements in practice in the United Kingdom. The only breathing system explained is the circle: although the reader is referred to our own *Physics for Anaesthetists* for details of the others, surely brief descriptions are warranted even in America. Alas, the statement that 'most patients given a general anaesthetic are paralysed' is probably not an Americanism.

Illustration is defintely a weak point: the photographs will date quickly and one black box with neatly coiled leads looks very much like another; however, I was endeared to the frankly messy picture of the nitrous oxide supply bank (complete with chalk letters on the wall; is SPL MIX for the boss or the technician?). The line drawings are a mixed bag, the few originals are excellent and those reprinted from elsewhere are mostly good but with a few horrors. The very first 'illustrates gas flow and mixing as well as the functional components encountered in a typical two-gas anesthesia machine'; indeed it does, but what is needed is a disentangled linear version of the information to aid comprehension, it seems particularly unfortunate that this drawing has climbed, upside down, onto the front cover of the book. More purpose-drawn diagrams with examples of artefacts as well as good informative output traces would add value in the next edition.

General sections on electrical safety and the philosophy of alarms would be useful additions. Serious omissions are descriptions of the design and purpose of peripheral nerve stimulators and gas scavenging systems and the place of helium, all of which are merely mentioned; one would also have liked to see the inclusion of oxygen concentrators and electromyography. The vanishingly brief chapter on drugs used in anaesthesia could vanish altogether, and this would reduce the size of the excellent index. The bibliography is also good.

The sturdy hard cover of this book should allow it to be chained to a comfortable chair in the anaesthetists assistants' den where it will be very useful to trainees and to new recruits to anaesthetics who should read it during those first few on-call spells when not being allowed to do very much intensifies the feeling of bewildered submersion in technology. Enthusiastic medical students and recovery room nurses will also enjoy it and established anaesthetists' assistants will doubtless sharpen their wits on it.

M.L. HEATH

Brief reviews by the Editor

The history of anaesthesia

Edited by R.S. ATKINSON AND T.B. BOULTON. Pp. 630. Royal Society of Medicine Services Ltd, London, 1989. £40.00.

This book is not the entire history of anaesthesia as its title misrepresents but the proceedings almost *in extenso* of the 2nd International Congress of the History of Anaesthesia. It is a moderately priced soft back volume but has taken a long time to appear since the meeting. Most, if not absolutely all, of those who claim an interest in this subject were at the meeting and have thereby excluded themselves as reviewers of the book: in any case, they all could purchase the book at considerably less than its current cost. The illustrations are a travesty of what can be produced by modern printing but apart from this serious detraction many of the essays are good bed-time reading: some are illuminating, a few amusing and many informative. The academic value of the book is lessened considerably by the absence of an index. The editors have, however, left their marks: careful and painstaking work and deserve our gratitude.

No fault compensation in medicine

Edited by R.D. MANN AND J. HAVARD. Pp. 273. Royal Society of Medicine, 1989. £20.00.

This book is also the proceedings of a meeting published by, and held at, the Royal Society of Medicine. However, the delay between meeting and publication in hardback is a mere 7 months (the contrast between this and the preceding

volume is obvious) and the production team is congratulated.

There are some chapters which should be read by everyone and some which may safely be passed over. One can confidently forecast that those by Diana Brahams, Chris Ham, Arnold Simanovitch, Arnold Morrison and Peter Carpenter will inform and provoke most anaesthetists. There are also 'verbatim' transcriptions of discussion which contain revealing statements from contributors who are not authors. It is a pity that so few practising doctors (and no anaesthetist) had their comments reported. The sole statement about anaesthesia is by a political surgeon and, because of the lack of a comma, his remark is meaningless. Once again there is no index (does the RSM not believe in them?) but despite this obvious deficiency the book contains sufficient information to make a permanent addition to a library essential.

Resuscitation of the newborn: Part 1—Basic Resuscitation, Part 2—Advanced Resuscitation

Two pamphlets have been prepared by a multidisciplinary working party. Copies are available from the Royal College of Obstetricians and Gynaecologists at £4.00 each or £7.00 for both parts. A wall chart on basic resuscitation of the newborn 'is available from the Department of Health, Leaflet Unit, Cannons Park, Government Buildings, Honeypot Lane, Stanmore, Middlesex HA7 1AY £2.40 plus postage'. The wall chart is to be used in conjunction with the pamphlet.

Anaesthesia, 1989, Volume 44, pages 1021–1025

Anaesthetic literature

This section of *Anaesthetic literature* contains references taken from *Current Contents—Life Sciences* for August 1989. The Journals which may be consulted when *Anaesthetic literature* is compiled are those listed in *Current Contents—Life Sciences*, published by the Institute of Scientific Information, Philadelphia, Pennsylvania, USA.

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The collator of this section is Dr L. Kaufman, MD, FFARCS, 145 Harley Street, London W1N 2DE. Dr Kaufman is prepared to provide on request, and at a modest charge, a new additional service to our readers. References from January 1984 have been entered on data base. The data are held on Dbase II, cpm 86 and on Dbase II, MsDos and are available on disc, together with a program providing search facilities. Enquiries direct to Dr Kaufman at the above address please.

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